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THE SPONTANEOUS HYPOGLYCEMIAS

IMPORTANCE OF ETIOLOGY IN DETERMINING TREATMENT

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ANN ARBOR, MICH.

In the past few years the general medical profession has gradually become aware of the existence of a clinical syndrome characterized by the periodic occurrence of spontaneous hypoglycemia with its resulting symptoms relieved dramatically by the administration of sugar. The association of hypoglycemia with an overdose of insulin and the concept of endogenous hyperinsulinism, proposed by Harris [1] in 1924 and established by Wilder and his co-workers [2] in 1927 have led to the widespread misconception that hyperinsulinism is synonymous with spontaneous hypoglycemia. To be sure, hyperinsulinism is one of the most important causes of periodic spontaneous hypoglycemia in man. But, as will be indicated, there are many other abnormalities which produce an abnormally low level of the blood sugar and which, consequently, produce the same train of symptoms. Thus, in any case in which it is suspected that symptoms are due to hypoglycemia, the presence of abnormally low levels of blood sugar must be demonstrated during attacks and the symptoms in question relieved promptly by the administration of sugar. This establishes only the nonspecific diagnosis of spontaneous hypoglycemia. The etiologic diagnosis must now be sought by every diagnostic method at our disposal, for on the etiologic diagnosis rests the indicated therapeutic procedure. It is evident, for example, that an exploratory operation, designed to discover a pancreatic insuloma, will be a useless and dangerous procedure when the hypoglycemia results from hypofunction of the pituitary or adrenal glands or when it is due to a disturbed glycogenic mechanism of the liver.

It is beyond the scope of this presentation to discuss individually each of the possible causes of spontaneous hypoglycemia. It is evident that many of these etiologic factors will appear infrequently in patients with proved spontaneous hypoglycemia. It can be calculated from Wilder's [23] figures of etiologic incidence that, of fifty-five patients with manifestations of hypoglycemia in whom fasting blood sugar levels of 60 mg. per hundred cubic centimeters or below were obtained, forty-one cases (75 per cent) were classified as falling into one of three groups, namely hyperinsulinism (organic type), neurosis (functional hyperinsulinism in our classification) and hepatic disease. Similarly, in an analysis of 278 cases of so-called spontaneous hypoglycemia reported by Martin and Hellmuth [24] only thirteen cases are found in which there were definite symptoms of hypoglycemia and in which fasting blood sugar levels below 60 mg. per hundred cubic centimeters were obtained. Ten of the thirteen cases (76 per cent) were listed as being due to organic hyperinsulinism, functional hyperinsulinism and liver disease. If one includes all the cases of functional hyperinsulinism regardless of the fasting blood sugar level [25] the figure becomes 88 per cent in Wilder's series and 83 per cent in that of Martin and Hellmuth.

ETIOLOGIC CLASSIFICATION OF SPONTANEOUS HYPOGLYCEMIA

I. Organic—recognizable anatomic lesion.
- (*a*) Hyperinsulinism.
  1. Pancreatic island cell carcinoma.[3]
  2. Pancreatic island cell adenoma.[4]
  3. Generalized hypertrophy and hyperplasia of islands of Langerhans.[5]
- (*b*) Hepatic disease.
  1. Ascending infectious cholangiolitis.[6]
  2. Toxic hepatitis.[7]
  3. Diffuse carcinomatosis.[8]
  4. Fatty degeneration.[9] "Fatty metamorphosis."[10]
  5. Glycogenosis (von Gierke's disease).[11]
- (*c*) Pituitary hypofunction (anterior lobe).
  1. Destructive lesions (chromophobe tumors, cysts and so on).[12]
  2. Atrophy and degeneration (Simmonds' disease).[13]
  3. Thyroid hypofunction (?secondary to pituitary hypofunction).[14]
- (*d*) Adrenal hypofunction (cortex).
  1. Idiopathic cortical atrophy.[15]
  2. Destructive infectious granulomas.[16]
  3. Destructive neoplasms.[17]
- (*e*) Central nervous system lesions (lesions of brain and brain stem; said to interfere with nervous control of blood sugar).[18]

II. Functional—no recognized anatomic lesion.
- (*a*) Hyperinsulinism [19] (?autonomic nervous system imbalance).
- (*b*) Renal glycosuria (severe degrees of low renal threshold for dextrose).[20]
- (*c*) Severe continuous muscular work.[21]
- (*d*) Pregnancy and lactation.[22]

From the Department of Internal Medicine, University of Michigan Medical School.

The expense of these studies was defrayed in part by a grant from the Horace H. Rackham and Mary A. Rackham Foundation.

Read before the Section on Pharmacology and Therapeutics at the Ninety-First Annual Session of the American Medical Association, New York, June 13, 1940.

Owing to lack of space, this article has been abbreviated here by the omission of bibliographic references and other matter. The complete article appears in the author's reprints.

25. Patients with functional hyperinsulinism, as a general rule, maintain normal fasting blood sugar values but respond excessively to insulogenic stimuli, which results in rapid and frequently profound depressions of the blood sugar level. It is the only type in which a clinical diagnosis of periodic spontaneous hypoglycemia is justified in the continued absence of abnormally low levels of the fasting blood sugar.

It appears, then, that from 80 to 90 per cent of all cases of spontaneous hypoglycemia fall into three groups, namely functional hyperinsulinism, organic hyperinsulinism and hepatic disease. With these points in mind, observations and procedures useful in differentiating the three important etiologic agents will be discussed. For purposes of clarity in differential diagnosis three illustrative cases are presented in the following order: (1) hepatogenic hypoglycemia, (2) organic hyperinsulinism and (3) functional hyperinsulinism:

CASE 1.—*Hepatogenic hypoglycemia.* This is one of six cases previously reported by us.[6] It represents a type of severe chronic hepatogenic hypoglycemia which can be improved to the point of clinical cure by surgical removal of the source of a hepatitis.

W. F., a white man aged 47, a laborer, had been in good health until March 1934, when he began to have periodic attacks of unconsciousness. These episodes always occurred between 3 and 7 a. m., which was from nine to twelve hours after the evening meal. The duration of an attack had varied from one to thirty-six hours. There had been an average of from two to three attacks each month.

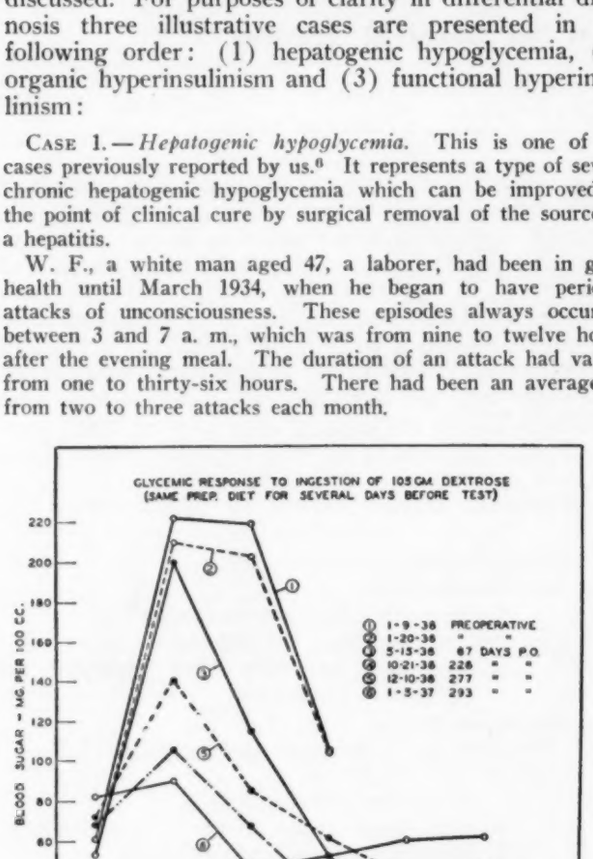
GLYCEMIC RESPONSE TO INGESTION OF 105 GM. DEXTROSE
(SAME PREP. DIET FOR SEVERAL DAYS BEFORE TEST)
BLOOD SUGAR - MG. PER 100 CC.
HOURS
1 1-9-38 PREOPERATIVE
2 1-20-38 " "
3 5-13-38 67 DAYS P.O.
4 10-21-38 226 " "
5 12-10-38 277 " "
6 1-5-37 293 " "

Chart 1 (case 1).—Hepatogenic hypoglycemia: Dextrose tolerance curves before and after cholecystectomy.

On Jan. 6, 1936, he was admitted to the University Hospital for study. Nothing of significance was found by physical examination. A dextrose tolerance test, after our standard preparatory diet,[26] gave the following response of the blood sugar: fasting 60 mg., one hour 220 mg., two hours 218 mg. and three hours 105 mg. per hundred cubic centimeters.

He was kept in bed and given a diet extremely low in carbohydrate (20 Gm. of carbohydrate, 50 Gm. of protein and 137 Gm. of fat). The following morning and for five successive mornings he was found disoriented and semicomatose or completely unconscious. The fasting blood sugar level each morning ranged from 14 to 18 mg. per hundred cubic centimeters. Prompt relief was obtained by administration of dextrose intravenously. One mg. of epinephrine subcutaneously was ineffective in relieving the attack or in raising the blood sugar significantly. No attacks occurred during the day.

Studies of the respiratory quotient[6] indicated no overcombustion of carbohydrate, as would be expected in hyperinsulinism.[27] Our attention was next turned to the liver, although there was no clinical suggestion of hepatic disease.

Evidence of marked impairment of liver function is shown by the following results:

1. Bromsulphalein retention 90 per cent and 70 per cent (blood bilirubin 0.25 mg. per hundred cubic centimeters).
2. Greatly impaired galactose tolerance.
3. Positive urinary urobilinogen test.
4. Abnormally low total serum proteins, with inversion of the albumin-globulin ratio.
5. Marked macrocytosis of the red blood cells with normal hemoglobin.

A cholecystogram showed slight visualization of the gallbladder with suspected stones. X-ray examination of the skull, the neurologic examination and basal metabolic readings gave no positive results.

On March 9, 1936, a cholecystectomy was done. The pancreas was examined carefully and found to be grossly normal. The liver was of normal size, abnormally pale and granular, with surface nodules about 2 mm. in diameter. The gallbladder was distended and contained several large stones and from 60 to 90 cc. of thick yellow pus. Biopsy of the liver showed active chronic cholangiolitis leading to early biliary cirrhosis.

Chart 1 demonstrates the gradual return of the glycogenic function of the liver in the postoperative period. Since we had shown that the normal quantity of absorbed dextrose was being removed by combustion, we were forced to conclude that the preoperative delay in clearing the blood of dextrose was due to faulty glycogenesis in the liver. That this function slowly returned to normal postoperatively is indicated by the gradually increasing speed of clearance from the blood of absorbed dextrose. Note that, as time passed, the fasting blood sugar level rose from 51 to 81 mg. per hundred cubic centimeters, the patient having been eating the same diet for several days before each test.

The level of the serum proteins gradually returned to normal with a reversion to the normal albumin-globulin ratio. Simultaneously the response to the bromsulphalein, galactose tolerance and urinary urobilinogen tests became normal. The erythrocytic macrocytosis disappeared completely.

For thirteen months after operation the patient, having returned to work, carried on his normal activities in apparent good health without any suggestion of hypoglycemia. On April 1, 1937, he died of acute coronary occlusion.

CASE 2.—*Organic hyperinsulinism.* This case[28] reported by us is unique in that four islet cell adenomas were removed at operation and complete cure of the hypoglycemia resulted. Continuous metabolic studies over a six months period yielded many data, proving among other things tremendous overcombustion of carbohydrate. Of 174 determinations of blood sugar in the preoperative period fifty-seven fell between 12 and 35 mg. per hundred cubic centimeters.

A. S., a woman aged 59, white, a widow, had been in excellent health until January 1930 (almost ten years before admission), when "attacks" began. While in a trolley car on her way home from work at 6:30 p. m., she was roused at the end of the line by the conductor, who told her that she had been staring straight ahead and jerking her right arm. She managed to change cars and arrived home in a dazed state, perspiring profusely and having only a vague recollection of what had transpired since she had left her office. She was ravenously hungry, recalling that she had had no breakfast and only a light lunch. She ate heartily and within fifteen minutes was again perfectly well.

From then on she noticed that the omission or sometimes the delay of a meal would bring on a "nervous spell." Throughout the ten year period attacks came at the average rate of one or two a week with no increasing frequency but with a tendency to become more severe. For the first seven years most of the attacks had occurred between 5 and 6 p. m., but more recently they had come almost always between 10 a. m. and noon. There had never been an attack during the night or before breakfast.

On Nov. 14, 1939, she entered the University Hospital for study. Except for obesity the physical examination was negative. She was given a general diet, and two days later at 11:45 a. m. a typical attack was observed. The blood sugar during the attack was 24 mg. per hundred cubic centimeters. The attack was quickly terminated by the intravenous administration of 5 Gm. of dextrose. After a five day period of standard dietary preparation the dextrose tolerance test gave the following results: fasting 37 mg., one hour 95 mg., two

hours 47 mg., three hours 45 mg., four hours 43 mg. and five hours 56 mg. per hundred cubic centimeters.

Liver function was normal as indicated by bromsulphalein, hippuric acid and galactose tolerance tests. Blood bilirubin, serum proteins and red blood cells were normal. A cholecystogram showed faint visualization with probable stone.

Skull roentgenograms showed a normal pituitary fossa. The visual fields were normal. The basal metabolism rate was minus 7 per cent. A sodium restriction test [29] showed no evidence of decreased function of the adrenal cortex.

On the provocative low carbohydrate diet, fasting blood sugar values ranged from 21 to 37 mg. per hundred cubic centimeters but, except for mild headache, there were no attacks before breakfast. Typical attacks came on daily, however, with clocklike regularity between 11:15 a. m. and noon with blood sugar values between 14 and 35 mg. per hundred cubic centimeters.

Respiratory data indicated marked overcombustion of carbohydrate when compared with normals under the same conditions.[28]

On Feb. 24, 1940, exploratory laparotomy was done. The liver was normal both grossly and microscopically (biopsy). It had a glycogen content of 5.5 per cent. At the junction of the distal and middle thirds of the tail of the pancreas a firm nodular area was felt. This area and all of the pancreas distal to it was resected and found to contain, deep in its substance, three separate and well encapsulated adenomas varying from 0.5 to 1.5 cm. in diameter. Sections revealed another of microscopic dimensions in the same region. The gallbladder, thickened and containing several pea sized stones, was removed.

Postoperatively a temporary period of hyperglycemia, glycosuria and ketonuria was observed. Over a period of two weeks the fasting blood sugar gradually became stabilized between 90 and 100 mg. per hundred cubic centimeters and subsequently remained there regardless of the diet (chart 2).

CASE 3.—*Functional hyperinsulinism.* This is the first case of three that we reported in introducing the use of a high protein diet in the treatment of this condition.

V. S., a girl aged 16 years, white, for two and one-half years had had periodic attacks of unconsciousness occurring once or twice a month. An attack usually began with visual disturbances followed by involuntary shaking of the extremities and irrational chattering. Unconsciousness frequently followed, lasting from five to fifteen minutes, sometimes associated with clonic convulsions. The attack would end rather abruptly, but mild disorientation and nausea remained for about an hour. The diagnosis elsewhere was epilepsy.

She was admitted to the University Hospital on Nov. 22, 1935. Physical examination revealed obesity without other abnormalities. The basal metabolic rate was minus 11 and minus 12 per cent. The visual fields and the pituitary fossa were normal. Dextrose tolerance tests gave the results seen in chart 3. No convulsion was observed in the hospital. However, when the blood sugar fell to 28 mg. per hundred cubic centimeters during a dextrose tolerance test she experienced the usual prodromal symptoms of an attack and said she was "about to have a fit," but none developed.

The fasting blood sugar level did not drop below 74 mg. per hundred cubic centimeters, regardless of the diet.

She was discharged with instructions to use a measured diet containing 110 Gm. of protein, 85 Gm. of carbohydrate and 135 Gm. of fat. In a six months follow-up period she experienced no convulsive attack. There had been, occasionally, however, the prodromal symptoms described.

DIFFERENTIAL DIAGNOSIS OF SPONTANEOUS HYPOGLYCEMIA

The Common Types.—When the possibility of hypoglycemia has not been kept in mind, a variety of erroneous diagnoses have been made. Among these are epilepsy, narcolepsy, intracranial tumor, diffuse disease of the brain or spinal cord, psychosis, psychoneurosis, neurasthenia and gastrointestinal disorders, particularly peptic ulcer.[30]

Granting the presence of an abnormally low value for blood sugar at a time when the patient is experiencing symptoms, and the prompt disappearance of those symptoms on the administration of sugar, how may one attempt to arrive at an etiologic diagnosis?

1. The Fasting Blood Sugar Level: When the previous diet has been normal, depression of the postabsorptive blood sugar value (taken before breakfast) below 50 mg. per hundred cubic centimeters means organic hypoglycemia with but few exceptions. This condition is present in cases 1 and 2 and absent in case 3. Provocative tests (restriction of carbohydrate or fasting) designed to disclose an abnormally low level of the fasting blood sugar must be interpreted with care. By means of a diet low in carbohydrate for very short periods of time we [31] have produced in normal people fasting levels ranging from 46 to 65 mg. per hundred cubic centimeters.[32] If, by provocative means, the level is found below 40 mg. per hundred cubic centimeters, one is justified in concluding that organic disease is present.

Functional hyperinsulinism, on the other hand, is not associated with low levels of the fasting blood sugar,

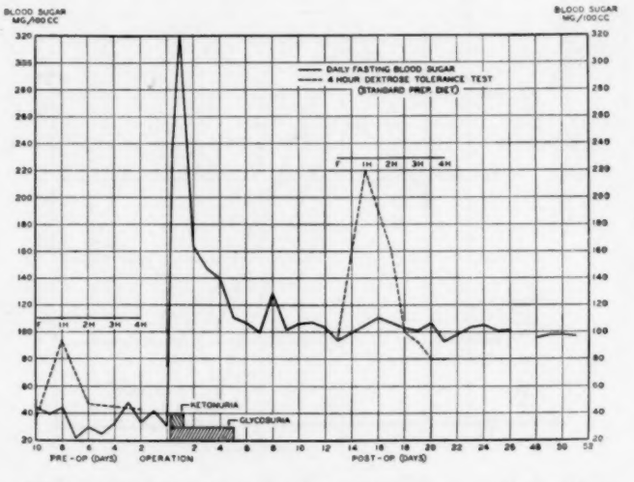

Chart 2 (case 2).—Organic hyperinsulinism: Preoperative and postoperative fasting blood sugar levels and dextrose tolerance curves.

depression of which by provocative tests is no greater than that produced in the normal individual. Thus the fasting blood sugar value is an aid in separating functional hyperinsulinism from the organic causes of spontaneous hypoglycemia.

2. The Dextrose Tolerance Test: Many writers have claimed that the dextrose tolerance test is of but little value in the differential diagnosis of the spontaneous hypoglycemias, citing as evidence the fact that all types of curves have been obtained in proved cases of organic hyperinsulinism; also that repeated tests in normal and diseased people show wide variations. We recognize the evidence but do not agree with the conclusions. It has been demonstrated repeatedly [33] that variations in the antecedent diet of the subjects affect profoundly the subsequent glycemic response to ingestion of dextrose. I [26] have proposed the use of a standard preparatory diet preceding the test. Wide variations are thus eliminated and much more comparable and reproducible curves result. Charts 4 and 5 show that in both organic and functional hyperinsulinism the character of the dextrose tolerance curve is significantly altered by variations in the carbohydrate content of the previous diet.

Of some diagnostic significance may be the fact that the loss of tolerance produced by previous diets low in carbohydrate is of much less intensity in organic hyperinsulinism than in functional hyperinsulinism. In the latter group the response approaches that which occurs in normal individuals.[26] In both organic and functional hyperinsulinism not associated with previous undernutrition or liver disease the dextrose tolerance response is characteristically of the low type. Except for the presence of abnormally low fasting levels in the organic type, the curve does not differentiate the two.

In hepatic disease, however, in which the glycogenic function of the liver is impaired, the glycemic response to ingestion of dextrose indicates a delay in clearing the blood of absorbed sugar in the usual length of time. This results in a high, plateau type of curve similar to that seen in diabetes mellitus, with the exception that the fasting level is usually abnormally low.[6] This type of response has been observed in experimental liver damage.[34] In association with other indications of

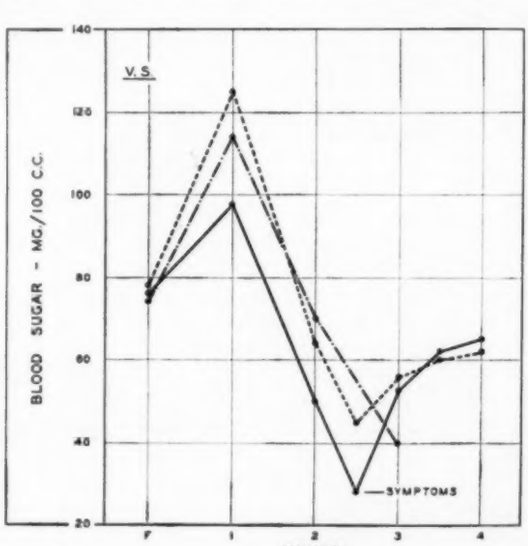

Chart 3 (case 3).—Functional hyperinsulinism: Repeated dextrose tolerance tests.

abnormal liver function (even though slight) this response is highly suggestive of the hepatogenic nature of the hypoglycemia.

3. Tests of Liver Function: Since it is acknowledged by all that one or several of the many functions of the liver may be severely damaged without proportionate impairment of other functions, it is imperative, in ruling out hepatogenic hypoglycemia, to test as many functions as possible. As a matter of routine we test for (1) bromsulphalein excretion, (2) urinary hippuric acid excretion (Quick test), (3) galactose tolerance, (4) urinary urobilinogen, (5) serum proteins (6) and make a hematologic study (? macrocytosis of red blood cells) and (7) a cholecystogram.

4. Clinical Course: In both of the main types of organic hypoglycemia (hyperinsulinism and liver disease) there is a marked tendency for progression of symptoms. Attacks become more frequent and severe. In liver disease the difficulty is one of an inability to maintain a normal level of the blood sugar during the overnight fast. Hence there occurs a gradual fall in the blood sugar, the lowest level being reached just before breakfast. Attacks occur usually in the early morning hours and are infrequent during the day. In organic hyperinsulinism, however, attacks of equal severity may occur from three to four hours after meals as well as during the night. This was seen in case 2, in which the level of blood sugar three hours after breakfast was frequently lower than the fasting level.

Functional hyperinsulinism exhibits no tendency to become more severe. There are periods of severity interspersed with periods of complete remission. Attacks almost never occur after midnight, the fasting blood sugar level usually being normal. Symptoms characteristically appear from three to four hours after meals, are associated with hypoglycemia and are relieved by food. The attacks are of short duration (from five to fifteen minutes) and always disappear spontaneously even though no food is taken.[35]

Less Common Types.—Three more well defined clinical states sometimes associated with hypoglycemia should be mentioned in the differential diagnosis. They are hypofunction of the anterior pituitary gland, hypofunction of the adrenal cortices and renal glycosuria. All three may be regarded as fasting hypoglycemias. The blood sugar is likely to be at its lowest level after the overnight fast. This is easily demonstrated in hypophysectomized or adrenalectomized animals and in phlorhizin diabetes, in which the renal threshold for dextrose is lowered. Clinically, hypoglycemic convulsions are extremely rare and institution of a diet high in carbohydrate is usually sufficient to control the hypoglycemia (see treatment).

The finding of glycosuria in a patient having attacks of spontaneous hypoglycemia does not establish renal glycosuria as the cause. In liver disease glycosuria alternating with hypoglycemia occurs.[6] A dextrose tolerance test will differentiate these conditions.

SURGICAL TREATMENT

1. *Indications for Exploration of the Pancreas.*—(*a*) Absence of extrapancreatic causes of hypoglycemia.

(*b*) Abnormally low level of blood sugar during symptoms and rapid relief of symptoms by the administration of dextrose.

(*c*) Repeated fasting blood sugar values below 50 mg. per hundred cubic centimeters when the patient has been eating an adequate diet.

(*d*) Depression of the fasting blood sugar below 40 mg. per hundred cubic centimeters by carbohydrate restriction.

If symptoms have been severe, progressive and of relatively short duration, especially in a subject in middle life or beyond, a malignant condition of islet tissue should be suspected and operation should not be delayed. Somewhat slower progression is observed in cases due to adenoma.

I[38] stated in 1936 that, "when an unquestioned clinical diagnosis has been made, there appears to be

35. Haist, Ridout and Best (Diet and the Insulin Content of Pancreas, Am. J. Physiol. **126**: P518 [July] 1939) have shown recently that animals deprived of food for seven days have much less insulin in their pancreas than have well fed animals. Since lack of carbohydrate in the previous diet results in decreased utilization of carbohydrate in normals (Sheldon, J. M.; Johnston, M. W., and Newburgh, L. H.: A Quantitative Study of the Oxidation of Glucose in Normal and Diabetic Men, J. Clin. Investigation **16**: 933-936 [Nov.] 1937. Sweeney,[32] Malmros,[33] Tolstoi,[33]), one is justified in assuming that this group (functional hyperinsulinism) responds to low carbohydrate diets by virtue of their depressing effect on insulogenesis. The periodic hypoglycemia experienced with normal diets must represent an increased responsiveness of the pancreas to the normal insulogenic stimulus. Thus the term "functional hyperinsulinism" seems justified. The good response to low carbohydrate feeding indicates that the islets respond to the usual stimuli which depress insulogenesis and are, thus, still under the usual controlling influences, but are excessively responsive. In organic hyperinsulinism production of insulin appears to be at least partially independent of depressing stimuli.

a 50 per cent chance that a pancreatic abnormality will be found at laparotomy." It seems from more recent statistics [28] that, with more rigid diagnostic criteria and more careful examination of the pancreas, an abnormality will be found in a much greater percentage (Whipple,[39] fifteen of eighteen cases in which operation was performed, 83 per cent).

When islet cell tumors have been found and removed, complete cure has resulted in the majority of cases. In several cases in which relief has not been obtained, a subsequent operation has revealed the presence of another adenoma, removal of which produced complete recovery. Many cases conforming roughly to the foregoing criteria have shown no apparent abnormality of the pancreas. In some of these a partial or subtotal pancreatectomy has been advised and done, with many disappointing results.[40] It must be recognized that the removal of a tiny adenoma has resulted in complete cure. Very small adenomas may be missed at operation. They may even be of microscopic dimensions (one of which was found in case 2). It is possible that very small tumors have been included in the resected pancreas of the cured cases and left behind in the uncured ones. A group of microscopic adenomas removed in a partial pancreatectomy could easily escape detection at pathologic examination unless many sections were made.

2. *Indications for Cholecystectomy.*—Excluding the usual indications for removal of the gallbladder, chronic hypoglycemia of hepatic origin may afford such an indication in selected cases. Case 1 is an excellent example of this situation. With no clinical suggestion of disease of the liver or of the gallbladder, the fasting hypoglycemia and the plateau type of dextrose tolerance curve (after standard dietary preparation) led to the finding of severely impaired liver function and the presence of active purulent cholecystitis. Removal of the source of the ascending hepatitis allowed a return of normal liver function and clinical cure of the hypoglycemic state. In contrast to chronic hepatic hypoglycemia due to progressive destructive lesions of the liver, which are universally fatal, this type can be helped greatly by removing the source of the hepatitis. Indications for operation are:

1. Postabsorptive hypoglycemia markedly intensified by restriction of dietary carbohydrate.
2. Postprandial hyperglycemia with intermittent glycosuria.
3. Hyperglycemic plateau type of dextrose tolerance curve with abnormally low fasting level.
4. Evidence of impaired function by other tests.
5. Evidence of a diseased gallbladder by cholecystography.
6. Presumptive elimination of other causes of chronic degenerative and destructive lesions of the liver.

MEDICAL MANAGEMENT

1. *Organic Hyperinsulinism.*—Medical treatment is notoriously unsuccessful in controlling this condition. When the diagnostic criteria that have been outlined have been met, operation should be urged since there is roughly a 75 per cent chance that a tumor will be found. When operation is refused it becomes necessary to feed a diet high in carbohydrate, giving meals as frequently as is required to prevent attacks. This leads to obesity and makes a subsequent operation more severe and postoperative complications more likely. Severity may progress to a point at which continuous intravenous dextrose is necessary to maintain the blood sugar above hypoglycemic levels.

Epinephrine: We [28] have found that the addition of epinephrine (1 mg. subcutaneously twice daily) to a dietary regimen which produced from three to four hypoglycemic convulsions daily prevented entirely the occurrence of attacks and kept the blood sugar above the attack level. In addition, we found that the rise in blood sugar produced by epinephrine was not associated with increased oxidation of carbohydrate. When a similar rise in blood sugar is produced by the ingestion of dextrose, a marked increase in the combustion of carbohydrate invariably occurs. The results are not surprising, since Cori and his co-workers,[41] Colwell and Bright [42] and others have found in animals that carbohydrate combustion is completely suppressed by the continuous injection of epinephrine. Our results with epinephrine in normal human beings afford similar conclusions.[43] It is probable that the subcutaneous administration of epinephrine in oil may be a valuable adjunct in the treatment of cases in which operation is refused or in those cases in which operation does not bring relief.

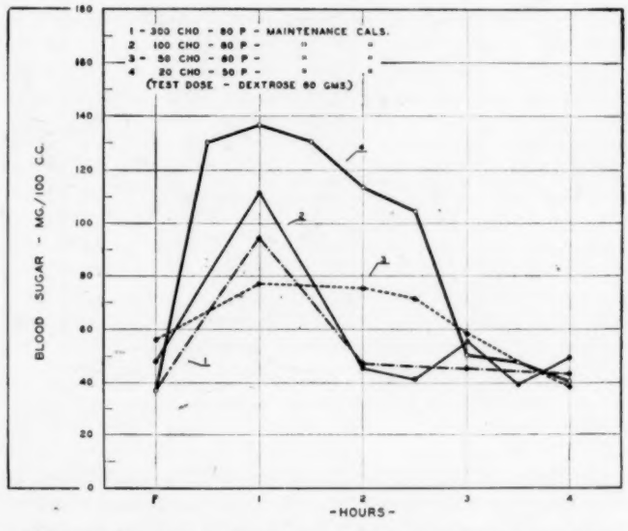

Chart 4.—Organic hyperinsulinism: Effect of previous diet on dextrose tolerance curve.

Ephedrine: The use of ephedrine has been only mildly effective and has failed to prevent attacks.

Adrenal Cortex Extracts: The possible effectiveness of this material in hyperinsulinism is suggested by the work of Jensen and Gratton [44] indicating the prevention of insulin convulsions in mice by previous administration of adrenal cortex extract and by the work of Britton and Sylvette,[45] Long [46] and others.[47] In case 2 the intramuscular administration of 30 cc. of adrenal cortex extract [48] daily (from four to five times the amount usually required to control Addison's disease) was ineffective in raising the blood sugar level and in preventing attacks. Simultaneous sodium and nitrogen balance studies indicated the potency of the extract used. It is likely that commercial extracts now available contain only small amounts of the "carbohydrate factors" (Kendall's fraction E and so on) of the adrenal cortex and that the future may bring better success with this form of therapy.

Anterior Pituitary Hormones: The experimental work of Young [49] and of Campbell and his co-workers [50] suggests that the use of insulin antagonists (diabetogenic and glycotropic factors) produced in the anterior pitui-

tary gland would be of benefit. Such extracts are not available commercially. Young [51] feels that diabetogenic hormone given in sufficient quantity to alter insulin production would cause complete destruction of all insular tissue and result in diabetes mellitus.

2. *Functional Hyperinsulinism.*—Into this group falls by far the greatest number of cases of spontaneous hypoglycemia, the characteristics of which have been

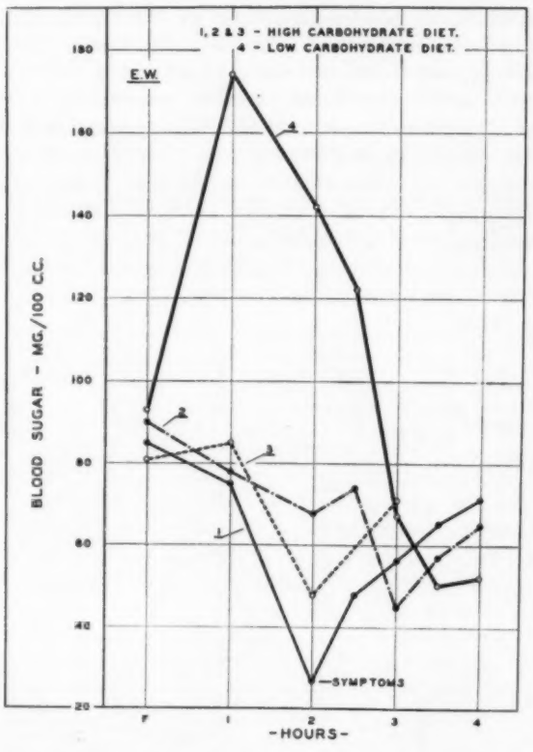

Chart 5.—Functional hyperinsulinism: Effect of antecedent diet on dextrose tolerance test.

described. Here the response to dietary regulation is usually good. Early in the development of diet therapy for these patients it was noted that diets high in carbohydrate resulted frequently in an increased number of attacks of greater severity. It was soon suggested that the postprandial rise of blood sugar associated with the ingestion of large amounts of easily absorbed carbohydrate afforded an added stimulus to insulogenesis with a resultant secondary abrupt fall of the blood sugar to hypoglycemic levels. Thus the oral administration of sugar would quickly alleviate a hypoglycemic episode but would be followed shortly thereafter by another attack, often more severe than the first. A similar situation can be produced in normal people [52] but it is of less intensity.

In an attempt, therefore, to prevent the postprandial rise in blood sugar with its subsequent secondary fall, Waters [53] proposed a frequent feeding schedule, using a diet low in carbohydrate and high in fat. On this regimen the patients were much improved but continued to have periodic attacks. John,[54] attempting to prevent the postprandial rise in blood sugar, gave small doses of insulin shortly after meals. He found that the secondary hypoglycemia could be prevented thus and reported good clinical results. In 1936 I [38] reported experimental and clinical evidence indicating that a diet high in protein and low in carbohydrate is beneficial in these cases and that such a diet could be given on a three meal schedule. We [55] had found that in both normal and diabetic persons the ingestion of large amounts of protein is followed by little or no rise in the blood sugar. Yet protein during its metabolism yields approximately 50 per cent of its weight as dextrose. When an equivalent amount of dextrose is ingested, as such or as carbohydrate food, a significant hyperglycemia is produced. Similar results were obtained in cases of functional hyperinsulinism, an example of which is shown in chart 6. The therapeutic effectiveness of the high protein diet is due to (1) the slow even rate at which dextrose is derived from protein, resulting therefore in no elevation of the blood sugar and consequently no secondary fall, and (2) the long period required for the absorption of protein and its conversion to dextrose, thus affording a slow steady supply of dextrose over a prolonged period of time.

We have had continued success with this type of treatment, with the occasional addition of a protein meal at bedtime. The average diet contains from 120 to 140 Gm. of protein, from 50 to 75 Gm. of carbohydrate and maintenance calories. Wilder [23] reports that at the Mayo Clinic best results have been obtained on a similar regimen, about 2 Gm. of protein per kilogram of body weight being prescribed.

3. *Hepatogenic Hypoglycemia.*—Except for cases of chronic hypoglycemia associated with infectious hepatitis in which surgical removal of the source of the hepatitis can be accomplished, treatment of hypoglycemia of hepatic origin is necessarily medical. In acute degenerative lesions of toxic origin the frequent or continuous administration both orally and parenterally of large amounts of carbohydrate serves a dual purpose: 1. It prevents the fasting hypoglycemia. 2. It aids in the regenerative process of the injured parenchymatous cells.[56] In progressive, chronic, destructive and degenerative lesions of the liver the attending hypoglycemia is controlled by the frequent administration of carbohydrate. A meal high in carbohydrate before retiring will frequently control the hypoglycemia found before

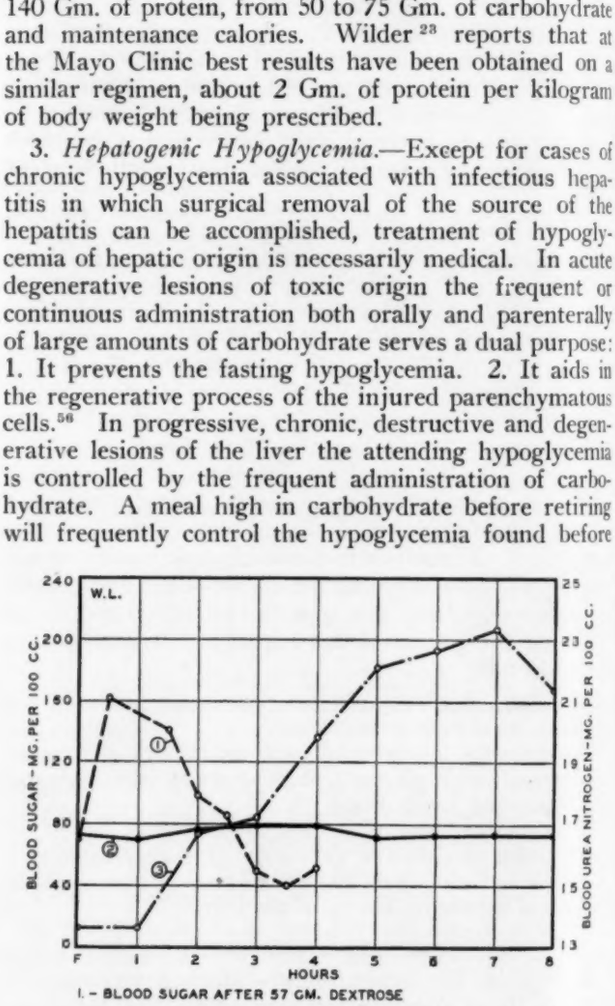

Chart 6.—Glycemic response to isoglucogenic quantities of dextrose and protein.

breakfast. In addition, the inclusion in the diet of foods high in protein is probably of value. Recent work [57] indicates a protective action on the liver of diets high in protein. The ability of the liver to deaminize and metabolize amino acids appears to be normal up to the very last stages of hepatic insufficiency.

4. *Pituitary and Adrenal Hypoglycemia and Renal Glycosuria.*—As noted before, the insufficiency in commercially available pituitary and adrenal extracts of the factors necessary to prevent insulin hypoglycemia eliminates the possibility of specific therapy at this time. Similarly there is no method available with which to raise the renal threshold for dextrose. A dietary schedule which includes frequent feedings high in carbohydrate is usually sufficient, however, to control the hypoglycemia.

ABSTRACT OF DISCUSSION

DR. HERBERT POLLACK, New York: Dr. Conn's work in the hypoglycemias has been appearing in the literature for about four years. It was Dr. Conn who, with Dr. Newburgh, first called attention to the use of the high protein diet in the therapy of the hypoglycemias. They pointed out that the carbohydrate available from the protein was liberated slowly over a period of time, thus preventing the drop in blood sugar during the night fast. It was this concept that led me to my employment of the high protein evening meal for the diabetic patient, using protamine zinc insulin. The nocturnal blood sugar curves of these patients resembled those of a patient with mild hypoglycemia. Dr. Conn made the statement that the rapidity of the blood sugar drop was important in the production of the hypoglycemic symptoms. This must be emphasized again. Daily one sees the reactions from protamine zinc insulin as being entirely different from those observed from the unmodified insulin, largely because the changes from the former are slow and from the latter rapid. I need not discuss at this point the possibility of certain of the symptoms of hypoglycemia such as trembling, tachycardia or blanching of the skin as being due to secondary discharge of epinephrine and other symptoms such as drowsiness, visual disturbances and coma as being due to hypoglycemia per se. The case illustrating the hepatogenic type of hypoglycemia is of especial interest to me. Dr. Lande and I reported three somewhat similar cases five years ago from a different point of view. We showed the diabetic type of dextrose tolerance curve associated with biliary tract disease which returned to normal postoperatively. Two of the patients had hypoglycemic episodes. We pointed out the caution necessary in using insulin postoperatively in certain "diabetic" patients after gallbladder surgery. Dr. Conn points out one of the significant differential points in the diagnosis of the etiology of hypoglycemia, that is the effect of the antecedent diet on the dextrose tolerance curve. This has been recognized for years in the so-called hunger diabetes. More important is the profound effect of low carbohydrate diets on the functional hypoglycemias as compared to the slight effect on the organic hypoglycemias. It is important to point out that the cause of hypoglycemia is the rapid drop after a rapid rise in blood sugar concentration. Samogi and Dolger and I have pointed out this exaggerated response in normal persons and in diabetic patients. We emphasized that many of the insulin shocks seen in diabetic patients occur after a period of hyperglycemia and glycosuria.

DR. E. J. KEPLER, Rochester, Minn.: It is important to differentiate hypoglycemia and hyperinsulinism. The former refers to the fact that the concentration of the blood sugar is subnormal, the latter to a specific form of hypoglycemia which results from an excessive production of insulin. Failure to make this distinction has been the cause of diagnostic and therapeutic errors. Recently there has been a tendency to diagnose "hyperinsulinism" on inadequate evidence. Disabling hyperinsulinism and spontaneous hypoglycemia are rare conditions. In contrast, however, there are many individuals who experience unpleasant sensations before mealtime. In some cases the concentration of the blood sugar may be found to be at low normal levels, 70 mg. per hundred cubic centimeters, for example. If such an individual happens to be hypersensitive or is neurotic, an erroneous diagnosis of "hyperinsulinism" frequently is made. Often this diagnosis precipitates an anxiety neurosis that becomes more of a problem than the symptoms for which medical advice was sought. In the main I agree with Dr. Conn's classification of the hypoglycemic states. In addition to those conditions which he has mentioned, we at the Mayo Clinic have encountered severe hypoglycemia of an indeterminate origin. In these cases severe hypoglycemic episodes continued to occur after practically all pancreatic tissue had been removed by one or more operations. Furthermore, in at least two instances with which I am familiar an adequate basis for the occurrence of the hypoglycemia could not be demonstrated at necropsy. This form of spontaneous hypoglycemia often is refractory to every known form of therapy; consciousness can be maintained only by the ingestion of food at intervals of four hours throughout the day and night, and eventually mental deterioration may occur, presumably because of repeated hypoglycemic insults to the central nervous system. It has become traditional to include hypopituitarism among the causes of hypoglycemia. Actually, hypoglycemic symptoms are rarely encountered in cases of anterior pituitary insufficiency. Subnormal concentrations of the blood sugar or flat dextrose tolerance curves may be obtained in cases of Simmonds' disease or chromophobe tumors of the pituitary body, but almost always the hypoglycemia is asymptomatic. The same remarks apply to the functional counterpart of anterior pituitary insufficiency; namely, anorexia nervosa.

DR. G. M. CHURUKIAN, Paris, Ill.: During the past two years I have done dextrose tolerance tests on patients complaining of dizziness, faint feeling, extreme nervousness and so on, exaggerated by high carbohydrate food intake. There were two types of patients: the obese diencephalic type and the asthenic type. Dextrose tolerance curves on the diencephalic type were close to the diabetic type in climbing high the first half hour. However, they manifested a sudden drop in one hour far below normal, to 40 or 50, while in the asthenic type the curves were of the flat type and usually below the base line. These patients were treated with high fat and high protein and low carbohydrate diet. The obese were given 125 Gm. of fat and from 60 to 70 Gm. of protein and the asthenic were given from 125 to 150 Gm. of fat and from 70 to 80 Gm. of protein. After three or four weeks the dextrose tolerance tests were rechecked. Almost all of these revealed more or less normal curves with relief of hypoglycemic symptoms.

DR. JEROME W. CONN, Ann Arbor, Mich.: The type of case to which Dr. Churukian refers probably belongs in the group which I have classified as functional hyperinsulinism. It should be stressed again that this type of transitory, symptomatic spontaneous hypoglycemia is the most common form encountered. It represents merely a more intense response to insulogenic stimulation than is exhibited by the average normal person. Thus the prevention of postprandial hyperglycemia in these "hyper-reactors" lessens the secondary, stimulative hypoglycemia which would otherwise occur. When a low carbohydrate diet intensifies the hypoglycemia, an etiology other than functional hyperinsulinism should be sought. Many errors in interpretation of the dextrose tolerance test will occur unless the subject has been eating a standard preparatory diet for at least three days before the test is done.

Curiosity May Be Disastrous.—Man's inherent and compelling curiosity, when well directed, has led to scores of important discoveries having far-reaching beneficial effects on man and beast alike, Dr. John R. Mohler, chief of the U. S. Bureau of Animal Industry, said in an address at the bicentennial celebration of the University of Pennsylvania. But curiosity about disease organisms may also result disastrously to man. When a stock raiser, for instance, finds a cow dead, apparently from anthrax, it is dangerous to open the animal's carcass in search of evidence of the disease. The body fluids of an animal dead from anthrax are full of dangerous bacilli, and a postmortem examination by one who does not know exactly what precautions to take may result fatally to the examiner. The U. S. Department of Agriculture warns emphatically against postmortem examination of animals that may have died from diseases dangerous to man, Dr. Mohler says. Leave this form of curiosity to veterinarians and stockmen experienced in this work.—Clip Sheet, U. S. Department of Agriculture, Oct. 20, 1940.

SERUM TREATMENT OF PNEUMOCOCCIC PNEUMONIA

GEORGE TAPLIN, M.D.
ROCHESTER, N. Y.

In July 1938, when the plans for this study of pneumonia were being formulated, two new therapeutic agents had just been reported. The first was unconcentrated antipneumococcus rabbit serum, reported by Horsfall, Goodner, MacLeod and Harris in May 1937.[1] The second was sulfapyridine, reported by Whitby on May 28, 1938.[2] At this time there had been no clinical experience with sulfapyridine in this country. On the other hand, there had been extensive experience with serotherapy for many years and the principles of treatment were well established.

In January 1938 the Bureau of Pneumonia Control of the New York State Department of Health supplied the Strong Memorial Hospital with nearly all the thirty-two types of therapeutic and all the thirty-two types of diagnostic rabbit serums for experimental purposes. Since the supply of horse serum of types I and V was ample and the results had been favorable in previous years, only patients sensitive to horse serum of these types were treated with rabbit serum in this series.

The preliminary results with the use of rabbit serum in the six month period January to July 1938 were favorable, so that when this study began rabbit serum was given preference over all other forms of specific treatment.

For the first eight months of this study (July 1938 to March 1939) no type III rabbit serum was available. Therefore, when sulfapyridine was supplied for experimental purposes by a commercial firm in the fall of 1938, it was felt that pneumonia due to the type III pneumococcus would provide an excellent test of its efficacy, since the mortality rate in the Strong Memorial Hospital for twelve years had averaged 45 per cent for lobar pneumonia caused by this type.

Because of the numerous complicating factors in pneumonia and the resulting difficulties in evaluating therapeutic measures, it was considered wise to use the new agents separately rather than in combination. Therefore, in this series all patients, with rare exceptions, received either serum or sulfapyridine therapy. Only those with the type III pneumococcus and those who were sensitive to serum or were allergic received sulfapyridine. The majority of patients were treated with either rabbit or horse serum. All patients received the same kind of general care and supportive measures as indicated.

This method of selecting cases simplifies and increases the accuracy of evaluating the results of specific therapy. Because there were only forty-three patients treated with sulfapyridine alone, it was decided to withhold the report on sulfapyridine until a later date, when more cases have been studied.

From the Department of Medicine, University of Rochester School of Medicine and Dentistry, and the Medical Clinics of the Strong Memorial and Rochester Municipal hospitals.

This report is part of a cooperative study of antipneumococcus serum made possible by the Bureau of Pneumonia Control of the New York State Department of Health, which has furnished the serums.

1. Horsfall, F. L., Jr.; Goodner, Kenneth; MacLeod, C. M., and Harris, A. H., Jr.: Antipneumococcus Rabbit Serum as a Therapeutic Agent in Lobar Pneumonia, J. A. M. A. **108**: 1483 (May 1) 1937.

2. Whitby, L. E. H.: Chemotherapy of Pneumococcal and Other Infections with 2-(*p*-Aminobenzenesulfonamido) Pyridine, Lancet **1**: 1210 (May 28) 1938.

OUTLINE OF THE PROGRAM OF STUDY

Selection of Patients.—All patients with pneumonia due to type II or higher types of the pneumococcus were treated with rabbit serum, with a few exceptions.

All patients with type III pneumonia due to the pneumococcus received sulfapyridine therapy.

All patients with pneumonia due to pneumococci of types I and V were treated with horse serum unless sensitive to horse serum.

Only exceptional patients received both serum and sulfapyridine. These are excluded from the present series unless they were given sulfapyridine first and failed to respond. A small group of infants who were treated with sulfapyridine first and failed to respond after forty-eight hours' trial were given serum; they are included in this report.

Routine Procedures for Serum-Treated Patients.—A complete history, physical examination, blood count, urinalysis and stool examination were made.

A history of allergy or of previous serum treatment was obtained.

Cutaneous and ophthalmic tests with normal horse and rabbit serums were performed.

Sputum was typed directly by the Neufeld quellung technic and cultured on a blood agar plate, and mouse inoculations were made in each case. A second specimen of sputum was retyped, and a pure culture of pneumococci was forwarded to the Health Bureau Laboratories in Albany, N. Y., for verification.

Blood was taken for culture prior to serum treatment, and repeated cultures were made daily while the patient was febrile.

X-ray examination of the chest was made in 90 per cent of the cases.

Routine determinations of the nonprotein nitrogen content of the blood, the plasma chlorides and the icterus index were made.

Special Studies.—Serum Antibodies: Three methods of determining the presence of circulating antibodies were used: (1) serum agglutinin titration, (2) determination of serum capsular-swelling antibodies and the (3) Francis skin test. All three tests were made in the majority of cases and were repeated at frequent intervals during the period of hospitalization.

Serum Complement Determinations: These tests were performed prior to treatment and repeated at frequent intervals in conjunction with the antibody studies.

PRINCIPLES OF TREATMENT AND CONTROL OF SERUM DOSAGE

In cases suitable for serum therapy as determined by a negative history of allergy and negative sensitivity reactions, a general plan of treatment was formulated in advance. In their original article on the use of unconcentrated rabbit serum, Horsfall, Goodner, MacLeod and Harris [1] stated that the average dose in their cases was 159 cc. In the beginning of this series this amount was used as a rough guide for initial doses. This was the amount of serum given in the average uncomplicated case, that is, to a patient with any type of pneumococcus other than type II, who was 40 years of age or less, and had involvement of only one lobe, a negative blood culture and disease of less than three days' duration. Larger initial doses were given in cases in which additional factors were present which are known to increase the mortality rate. These include age above 40, type II pneumococcus infections, involve-

ment of more than one lobe, alcoholism, pregnancy and duration of the disease of more than three days. In addition, patients presenting marked toxicity and a positive reaction to the urine precipitin test were treated with a projected dose as large as that which would have been given in the presence of bacteremia (see later portion of section). This was done because, as Cruickshank [3] has shown, the disease in cases presenting such a picture is more severe and the mortality rate is many times higher than in similar cases in which the precipitin test is negative.

Subsequent dosage was guided by two main factors, namely clinical response and development of excess demonstrable serum antibodies. The rationale for considering the factor of excess antibody is based on the experiments of Robertson,[4] who showed that for the resolution of the pneumonic process the macrophages, which are largely responsible, require the presence of specific antibody for phagocytosis, just as do the polymorphonuclear cells in the early stages of the disease. It has been shown that, once an excess serum titer has been established, it persists for from seven to fourteen days. Observations made in series verify this work.

In cases in which good clinical response occurs to the projected dose it is important to determine whether excess serum antibody is present. If it is, further serum treatment is superfluous. If there is no demonstrable serum antibody or if the reaction to the Francis test is negative, it is likely that without additional serum treatment a relapse or slow resolution will follow. Therefore, in cases in which there was a good clinical response but demonstrable serum antibodies failed to develop, additional serum was given until a definite level of antibody was present. Generally this required only small doses of serum, from 20 to 60 cc.

Cases in which there was no response to the projected dose of serum after twelve to eighteen hours were studied in an attempt to determine the cause of serum failure. These additional points were checked in each case at this time:

Blood Culture.—Patients with bacteremia were given additional serum, even when the clinical response was favorable.

Sputum Typing.—Retyping the sputum provided the answer to apparent serum failure in several cases, especially in those in which more than one type of pneumococcus was isolated from the sputum. Culture of the sputum was valuable in the diagnosis of cases of pneumonia due to Staphylococcus aureus, a hemolytic streptococcus and Friedländer's bacilli, in which a pneumococcus was found in the initial typing by the Neufeld technic.

Physical Examination.—The most common physical manifestations causing apparent serum failure were pleural effusion and spread of the process to another lobe. No cases of empyema were seen in this series.

Serum Antibody Study and Francis Skin Test.—These tests were of about equal value in guiding subsequent dosage. In several cases of initial serum failure, inadequate dosage was found to be the cause. This was determined by the presence of a negative blood culture, failure to demonstrate serum antibodies or a negative Francis skin test, verified typing of the sputum and elimination of physical causes for serum failure.

Blood Complement Determination.—The value of blood complement determination appeared in cases in which there was no cause for serum failure after the aforementioned points had been checked. In this series, six patients who failed to respond to adequate and specific serum therapy were found to be deficient in serum complement. These patients were given fresh normal human serum in 200 to 400 cc. doses and responded by crisis within twenty-four hours. It is believed that further study of this factor in pneumonia and other infectious diseases may prove fruitful. The rationale for this feature of treatment is that complement speeds the rate of phagocytosis of sensitized bacteria.

TECHNIC OF SERUM ADMINISTRATION

Prior to intravenous therapy, sensitivity tests—cutaneous, ophthalmic and intravenous—were performed on all patients according to the methods used by Horsfall, Goodner, MacLeod and Harris.[1] Undiluted serum was administered by the syringe method in 40 to 100 cc. doses at the rate of 2 to 4 cc. a minute. The first therapeutic dose was usually 40 cc. or less. If no reaction occurred after such a dose, the rest of the projected amount of serum was given in the next dose unless this amount was more than 120 cc. In such instances two or more injections were given at one to two hour intervals.

TESTS USED IN THE CONTROL OF SERUM DOSAGE

1. *Francis Skin Test.*—Purified pneumococcus polysaccharide substance was supplied by the New York State Laboratories for types I, II, V, VII and VIII. The test was performed prior to treatment in all cases because it has been shown [5] that a definite group of patients give a false positive reaction: in such cases the test is of no value. A positive reaction consists of an area of erythema from 2 to 5 cm. in diameter plus the formation of a definite wheal at the site of injection appearing during the first hour after injection, and usually within twenty to thirty minutes. In some cases the reaction became positive after the first or second therapeutic dose of serum. In general, a positive Francis cutaneous reaction coincided with the development of excess serum antibodies. In a few cases positive cutaneous reactions never developed, although agglutinins were present in the serum and recovery occurred. However, in no case were a positive reaction to the Francis test and a negative agglutination reaction found. Therefore, a Francis skin test negative before treatment becoming positive after treatment is a reliable test for the presence of excess circulating antibody in cases of types I, II, V, VII and VIII pneumonia.

2. *Serum Antibodies.*—Serum agglutinin titrations and capsular swelling antibody determinations were done in a large percentage of cases. These two tests were run simultaneously in the following manner:

An autogenous antigen was prepared from each patient. The peritoneum of a mouse was inoculated with the patient's sputum, and the exudate was aspirated within four to twelve hours. This exudate was sus-

3. Cruickshank, Robert: Urinary Excretion of Pneumococcus Polysaccharide in Lobar Pneumonia, J. Path. & Bact. **46**: 67 (Jan.) 1938.

4. Robertson, O. H.: Recent Studies on Experimental Lobar Pneumonia; Pathogenesis, Recovery and Immunity, J. A. M. A. **111**: 1432 (Oct. 15) 1938.

5. MacLeod, C. M.; Hoagland, C. L., and Beeson, P. B.: The Use of the Skin Test with the Type Specific Polysaccharides in the Control of Serum Dosage in Pneumococcal Pneumonia, J. Clin. Investigation **17**: 739 (Nov.) 1938.

pended in saline solution and centrifuged at low speed for two to three minutes to throw down the white blood cells and débris. The supernatant fluid containing pneumococci was then stained with methylene blue and kept in air tight bottles. The agglutination and capsular swelling tests were done simultaneously by the slide method. A drop of the patient's serum, obtained by venipuncture, was mixed with a drop of the stained suspension of pneumococci. The mixture was incubated at 37 C. for one-half hour and read. Three additional dilutions of serum, 1:5, 1:10 and 1:20, were prepared and mixed with the antigen, incubated and read, as in the case of the undiluted serum. When the patient's serum had a considerable excess of antibody, the agglutination and capsular swelling reactions occurred almost immediately. It was found that either an agglutinin or a quellung titer of 1:20 or more was an indication of sufficient serum therapy. Excess antibodies determined by this method persisted generally for at least ten days, and in some cases as long as a month. This technic of serum antibody determination has the advantage of simplicity over the macroscopic agglutination method and is accurate enough for practical purposes.

Comparison of Results of Treatment of Pneumonia with Rabbit and with Horse Serums *

Critical Factors	Rabbit Serum	Horse Serum
Number of cases	65	35
Mortality	2 cases (3%)	3 cases (8.5%)
Average age	45.2 yr. (exclusive of 5 infants)	44.4 yr.
Lobes involved	1 lobe, 41 cases 2 lobes, 22 cases 3 lobes, 2 cases 4 lobes, 0 case	1 lobe, 23 cases 2 lobes, 10 cases 3 lobes, 1 case 4 lobes, 1 case
Day of disease treatment was started	1st day, 28 cases 2d day, 16 cases 3d day, 7 cases 4th day, 7 cases 5th day, 3 cases 6th day, 3 cases 7th day, 1 case	1st day, 8 cases 2d day, 13 cases 3d day, 2 cases 4th day, 5 cases 5th day, 1 case 6th day, 5 cases 10th day, 1 case
Incidence of bacteremia	9 cases (13.8%)	10 cases (28%)
Average dose of serum	186 cc.	133 cc.
Incidence of alcoholism	7 cases (10.7%)	6 cases (17.1%)
Incidence of heart disease	12 cases (18.4%)	6 cases (17.1%)
Immediate serum reactions	8 cases (12.3%)	16 cases (45.7%)
Incidence of serum disease	20 cases (12.3%)	15 cases (42%)
Clinical response to serum treatment		
Crisis 12 hr.	31 cases	17 cases
Crisis 24 hr.	18 cases	7 cases
Crisis 48 hr.	6 cases	3 cases
None	1 case	2 cases
Lysis	9 cases	5 cases
Incidence by type of organism		
Type I	8 cases	18 cases
Type II	8 cases	1 case
Type IV	0 case	3 cases
Type V	6 cases	1 case
Type VI	1 case	1 case
Type VII	8 cases	5 cases
Type VIII	5 cases	3 cases
Type XIV	4 cases	1 case
Type XIX	.....	1 case
Higher types	25 cases	

* One cc. of concentrated horse serum contains 2,500 units; 1 cc. of unconcentrated rabbit serum contains approximately 2,000 units.

3. Blood Complement Determination.—Blood complement was measured by the serial dilution method, as used by the New York State Laboratories in Wassermann determinations. It was found that the amount of serum of normal persons necessary to hemolyze 0.2 cc. of a 2.5 per cent suspension of sheep cells incubated at 37 C. in the water bath for fifteen minutes was 0.01 to 0.02 cc. In the cases of pneumonia dilutions containing 0.01 to 0.05 cc. of the patient's serum were used in the aforementioned manner and read. The end point was that dilution which gave complete hemolysis. Fresh sensitized sheep cells were obtained daily from the Wassermann laboratory. All tests were performed within two hours after the specimens had been obtained.

RESULTS OF TREATMENT WITH HORSE SERUM

Thirty-five patients were treated with concentrated horse serum supplied by the New York State Department of Health, in the same manner and with the same controls as the series of sixty-five patients given rabbit serum. There were three deaths. The relative response to treatment, the serum reactions, the dosage and other factors observed in this series are compared in the accompanying table with similar observations for the rabbit serum treated patients.

COMMENT

From the data recorded in this series of 100 cases of proved, typed pneumococcic pneumonia, it appears that both antipneumococcus horse and rabbit serums are valuable therapeutic agents. The number of cases is too small to form a basis for definite conclusions as to the relative curative value of the two agents. However, it appears so far that unconcentrated rabbit serum is superior in regard to its relative freedom from reactions. There was a collapse reaction in only one of sixty-five cases. The patient responded to treatment with appropriate measures. The frequency of febrile reactions was almost four times less with unconcentrated rabbit serum than with horse serum. Of the sixty-five cases in which rabbit serum was used, severe serum disease developed in only four, and in sixteen cases the disease was mild, lasting only one to three days and responding readily to symptomatic treatment.

No control cases were used in this series. However, prior to 1938 specific therapy was available only for pneumonia of types I and II, and the data for these and other types have been gathered and interpreted by Dr. Sidney Rothbard and Miss Priscilla Cummings, of these hospitals. The gross mortality for pneumococcic lobar pneumonia for 1926 to 1936 was 40.5 per cent. This included 111 cases of type I pneumonia in which horse serum was employed, with a mortality rate of 26 per cent. When the cases of type III pneumonia were excluded, the mortality rate in their series of 645 cases was still 38 per cent.

Therefore, it is felt that the mortality rate of 5 per cent in this series, cases of type III in which sulfapyridine was used being excluded, represents the effect of adequately controlled serum treatment. It is realized that the incidence of bacteremia of 19 per cent is slightly lower than in previous years (the average for 1926 to 1936 being 24 per cent) and probably contributes to the low mortality rate. Even under such circumstances the other factors present in this series, such as an average age of 45, multiple lobe involvement in 36 per cent of the cases, heart disease in 18 per cent and alcoholism in 13 per cent, should have produced a mortality rate of 30 per cent or more if specific treatment had been withheld. Therefore it is felt that serum treatment has reduced the mortality rate at least sixfold and has not been responsible for a single death.

SUMMARY OF THE FIVE FATAL CASES

CASE 1.—R. B., a man aged 52, known to be chronically alcoholic, entered the hospital on the tenth day of type I lobar pneumonia, which involved the entire right lung. He was in desperate condition, with extreme cyanosis, dyspnea and gross distention. He was treated with 350,000 units of type I horse serum during the first thirty-six hours after his admission.

His temperature returned to normal in eighteen hours, and excess antibodies were demonstrated in the blood serum. Repeated blood cultures were sterile, but the patient remained ill. Distention and delirium were the main therapeutic problems. The distention was finally controlled by intravenous injection of saline and dextrose solution, prostigmine, enemas, stupes and Wangensteen suction. The delirium failed to respond to intravenous administration of dextrose, saline solution, sedatives, and large doses of vitamin B_1.

Twenty-four hours before death the temperature, which had been normal for five days, became elevated, and sputum on retyping revealed pneumococcus types VIII and XIX, in addition to the original type I. Rabbit serum treatment for the higher types was withheld because there was no spread of the pneumonia and it was thought that the fever might be due to beginning serum disease. One hour before death the patient became comatose and slight stiffness of the neck developed. Lumbar puncture revealed sterile fluid under moderately increased pressure. A blood culture taken at this time revealed type VIII pneumococci. Postmortem punctures of the lung revealed the same organism. Permission for autopsy was refused.

This is the type of case in which there might well have been a favorable response to the combined use of serum and sulfapyridine.

CASE 2.—F. T., a man aged 64, entered the hospital on May 4, 1939, with congestive heart failure due to hypertensive and arteriosclerotic heart disease. He responded well to digitalis and diuretics. On May 10 fever, cough, bloody sputum and pain developed in the left side of the chest. Twenty-four hours later he had signs of pneumonia in the lower portion of the left lung, and type I pneumococci were found in the sputum. A blood culture taken on May 12 yielded type I pnuemococci. On the same day he received 300,000 units of type I horse serum in five doses. The vital signs, except for the pulse rate, returned to normal in twenty-four hours. A blood culture taken on May 13 was sterile. The patient became delirious, circulatory collapse developed and he died four days after the onset of the pneumonia. Determinations of blood antibodies were not made, but the Francis test remained repeatedly negative. Autopsy revealed type I pneumonia in the lower lobe of the left lung, early empyema and arteriosclerotic heart disease with congestive failure.

In retrospect, the cause of continued toxicity after serum treatment was the empyema, which must have developed on the second or third day of the pneumonia. This also explains the persistently negative reaction to the Francis test.

CASE 3.—C. N., a man aged 26, known to be epileptic, was admitted to the hospital on the sixth day of type I pneumonia, which involved the entire right lung and the lower lobe of the left lung. A total of 450,000 units of horse serum was administered during the thirty-six hour period after admission. Repeated blood cultures were sterile. There were no serum reactions. One hour after the first dose of serum the patient had a severe epileptic convulsion, lasting thirty minutes, which was followed by pulmonary edema. Symptomatic measures and sulfapyridine failed to alter the course of the disease. The patient died on the eleventh day of the disease with involvement of all five lobes of the lungs and pulmonary edema.

CASE 4.—C. B., a man aged 48, known to be chronically alcoholic, with rheumatic heart disease of class 2 b, was admitted to the hospital twenty-four hours after the onset of type II pneumonia, which involved the lower lobe of the right lung. On admission the blood culture yielded 10 colonies per cubic centimeter, and the initial white blood cell count was 7,600. He was given 200 cc. of rabbit serum in four doses on the day of admission. The vital signs, except for the pulse rate, returned to normal in twelve hours, and the blood stream was sterile. Congestive heart failure, auricular fibrillation and delirium tremens developed. In spite of oxygen therapy and digitalization, the patient became comatose and died thirty-six hours after admission.

Autopsy showed lobar pneumonia, type II, in the lower lobe of the right lung, rheumatic heart disease with congestive failure, hemorrhagic gastritis, and sterile pleural effusion on the right. A postmortem culture of the blood was sterile.

This patient should have been digitalized on admission, before signs of heart failure had developed.

CASE 5.—A. H., a man aged 52, with chronic alcoholism, entered the hospital on the sixth day of type V pneumonia, which involved the lower lobes of both lungs. Repeated blood cultures were all sterile. On admission he presented toxicity, deep jaundice, distention and delirium. He was treated with 320 cc. of type V rabbit serum given at two hour intervals in five doses. He responded by crisis in twenty-four hours. Two days later, in spite of a high caloric diet, intravenous injection of dextrose and large doses of vitamin B_1, he had a recurrence of the delirium. The delirium failed to respond to the aforementioned measures plus oxygen inhalations and sedation, and the patient became comatose and died four days after admission. Autopsy revealed lobar pneumonia, type V, in the lower lobes of both lungs, early cirrhosis of the liver, toxic hepatitis, pericardial effusion, dilatation of the right side of the heart and cerebral edema.

Of all the complicating factors noted in this series, the most serious and difficult to treat was alcoholic delirium. In three of the five fatal cases death was mainly due to this factor.

SUMMARY AND CONCLUSIONS

One hundred patients with twenty-three proved types of pneumococcic pneumonia were treated with serum plus the usual supportive measures. There was a gross mortality rate of 5 per cent. Rabbit serum was preferred because it caused considerably less immediate reaction than horse serum. On the basis of comparison of statistics for this and previous years, both rabbit and horse serum are considered effective and valuable therapeutic agents.

As a result of this study plus experience gained with the use of sulfapyridine in cases of type III pneumonia and serum sensitive cases of other types, it is felt that the program and principles of treatment set forth in this paper should be continued during the coming year, but that certain modifications are needed. These modifications are related to the place of serum and sulfapyridine or their combination in the treatment of pneumonia. The tentative plan for the year 1939-1940 is as follows:

1. All patients with type III pneumonia are to be given sulfapyridine unless a contraindication exists. If these patients fail to respond in twenty-four hours, type III rabbit serum is to be administered in addition.

2. Patients with early stages of pneumonia due to pneumococci of types I, II, V, VII, VIII and XIV are to receive serum, preferably rabbit, when the etiologic diagnosis can be made without delay. Patients who fail to respond in twenty-four hours are to be given sulfapyridine.

3. Treatment of all patients with types of the pneumococcus higher than XIV is to be started with sulfapyridine, and serum is to be given in addition only when necessary.

4. Allergic and serum sensitive patients are to be given chemotherapy.

5. Postoperative patients for whom nausea and vomiting would be dangerous are to receive serum therapy.

6. Patients with multiple types of pneumococci are to be given sulfapyridine first and serum later if necessary, except when one of the organisms is of type I or II.

7. Severely ill patients for whom the outlook is obviously poor are to receive sulfapyridine for the first twelve hours and then specific serum.

It is felt that these modifications should reduce further the mortality rate from pneumococcic pneumonia.

THE USE OF THE URINE IN THE CHEMICAL TEST FOR INTOXICATION

POSSIBLE ERRORS AND THEIR AVOIDANCE

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Determination of the amount of alcohol in the blood has now become an important feature in the medicolegal diagnosis of intoxication. It is usually impossible, however, to obtain blood for direct analysis; if it is obtained, the findings may be excluded as evidence in court.[1] Consequently an indirect determination of the concentration is now often made from saliva, from lung air and especially from urine.[2]

The use of the urine has some practical advantages. Under properly controlled conditions—but only under such conditions—the concentration of alcohol in the urine is an accurate index of the concentration of alcohol present in the blood at the time the urine is obtained. As the test is now often carried out by or for the police, serious discrepancies may occasionally occur, and the concentration of alcohol in the urine may differ widely from that in the blood. Consequently this testimony may jeopardize the individual who is suspected of being intoxicated and this useful test may be finally discredited.

The concentration of alcohol in the urine as secreted is known to correspond exactly to that simultaneously in the blood in the relation of the solubility of alcohol in the two fluids.[3] But the concentration in the urine that has accumulated in the bladder may not show this correspondence. Except for short periods the concentration of alcohol in the blood is not constant but instead it rises or falls, depending on whether the rate of absorption of alcohol from the alimentary tract is greater or less than the rate of oxidation and elimination. The concentration of alcohol in the urine secreted varies correspondingly. Consequently the urine that collects in the bladder is a composite.

This fact has been recognized but the very considerable differences in the concentration of alcohol in urine and blood, which are shown here, have not previously been demonstrated. Attorneys have pointed out in court that the concentration of alcohol in bladder urine collected over an undetermined length of time might be higher than that in the blood at the time the test was made. Medical experts, while admitting this fact, have countered with the statement that the concentration in bladder urine represents an average which is therefore lower than the highest concentration reached in the blood.

If the only fact in question is whether or not the suspect has taken alcohol, the presence of alcohol in the urine gives a positive answer. But the present tendency is to judge the extent of intoxication from the concentration of alcohol in the urine. It is here that an important distinction must be made. It is only the concentration of alcohol in the blood that is the significant value; the sole purpose of the test on the urine is to obtain this value. The concentration of alcohol in the blood, when determined directly on the blood or indirectly from the lung air, is the concentration which exists exactly at the time the test is made. But when the concentration in the blood is estimated indirectly from the urine the figure obtained may be that for a concentration which existed at some indeterminable time previously. It may be considerably higher than the concentration in the blood. The suspect may thus be penalized accordingly.

The suspect in whom a high concentration of alcohol is found in the urine, regardless of the length of time the urine has been retained, probably deserves conviction. But laboratory tests should not indicate mere probabilities. Precision is important in view of the standards embodied in the laws of some of our states[2] and advocated by a committee of the American Medical Association[4] and by the National Safety Council.[2] The essential features of the standard now most generally in favor are that a concentration of 0.05 per cent or below in the blood shall constitute conclusive evidence of sobriety; 0.15 per cent or above, evidence of intoxication, and values between these extremes probability of intoxication. The fact that there is a considerable range as between the concentration of 0.05 and 0.15 per cent does not lessen the need for precision, since a slight variation near either of these values may place the accused incorrectly in one or another of the categories arbitrarily defined. Moreover, the establishment of any quantitative value for the concentration of alcohol in the blood presupposes that it shall be the concentration at the time the test is made.

The data given in the accompanying table illustrate the influence of the frequency of voiding on the concentration of alcohol present in the urine as compared to that in the blood. Eleven male subjects ranging in age from 22 to 54 years and in weight from 122 to 187 pounds (55 to 85 Kg.) were given in each test 250 cc. of undiluted whisky. The concentration of alcohol in the blood was determined at one-half hour intervals directly from the capillary blood and indirectly from the lung air.[5] The concentration in the urine was determined at intervals ranging from thirty minutes to six hours; no urine was voided between these intervals. All the values given are averages for the eleven subjects; each column presents the results from a separate series of experiments. The concentrations of alcohol as obtained from the urine are converted to those of blood on the basis of a coefficient of 1 : 1.3.

During the rapid absorption of alcohol from the alimentary tract the concentration in the blood, as determined from the urine, is low; this is true even when, as here, the bladder is emptied of urine at the time the alcohol is ingested. Thus the urine voided at thirty minutes and one hour after the ingestion of the alcohol gave values (column 3 in the table) of 0.037 and 0.163 per cent; the concentrations in the blood (column 2) were 0.152 and 0.193 per cent. These differences can be explained from the facts that (1) the alcohol in the urine secreted during the first half hour is diluted with the urine retained in the pelvis of the kidney and (2) the rise in concentration of alcohol in the blood during

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the first half hour after ingestion is rapid. It rises from zero to nearly the maximum concentration attained; the composite concentration in the urine thus falls between extreme values and may therefore be considerably lower than the higher of these values.

After the initial rise during active absorption of alcohol, the concentration in the blood falls only as alcohol is oxidized and eliminated. This fall is slow. For the amount of alcohol given here, ten hours elapsed between the maximum concentration of alcohol in the blood, 0.193 per cent, and the disappearance of the alcohol. The average fall in the concentration as measured was therefore 0.0096 per cent for half an hour; the extremes found were 0.002 and 0.019 per cent. Over any short period of time, such as one-half hour, the concentration of alcohol in the urine collected in the bladder lies between very narrow extremes.

The close correlation of the concentration in the blood as measured directly and as obtained from the urine at half hour intervals is shown in columns 2 and 3 of the table. Between one and one-half and ten hours after the consumption of the alcohol the concentrations obtained at half hour intervals on the blood gave an average value of 0.103 per cent and from the urine 0.108 per cent; the maximum deviation at any single measurement was 0.009 per cent.

When the time over which the urine is collected is increased, the correlation becomes progressively less precise. Thus when the urine was collected for intervals of one hour the average values were 0.087 and 0.098 per cent and the maximum deviation for any single measurement 0.019 per cent. When collected for two hour intervals the average values were 0.087 and 0.106 per cent and the maximum deviation 0.042 per cent. For four hour intervals the average values were 0.055 and 0.098 per cent and the maximum deviation 0.052 per cent. Thus when urine has collected for a period greater than one hour at the time the test is made an appreciable error may be introduced into the determination made from the urine.

When the urine is collected for long periods of time the error assumes such magnitude as to invalidate any quantitative conclusion. Thus when the subjects did not void for six hours after ingestion of alcohol, column 9, the concentration as estimated from the urine was 0.158 per cent but that determined on the blood was 0.078 per cent. When they voided at one hour and at eight hours, column 10, the concentrations at the latter time were respectively 0.150 and 0.037 per cent. It is important with regard to the procedure to be suggested here for obtaining accurate values from the urine that the samples of urine taken one-half hour after those which gave the values of 0.158 and 0.150 per cent, columns 9 and 10, were 0.069 and 0.034 per cent; the corresponding concentrations as found in the blood at this time were 0.066 and 0.030 per cent.

In addition to the experiments recorded in the table, one subject was given 250 cc. of whisky on going to bed at 10 p. m.; the urine was retained until 8 a. m. At this time the concentration in the blood, as judged from the urine, was 0.11 per cent. Direct determination on the blood showed no alcohol.

Urine does not ordinarily accumulate in the bladder for six hours or more as in some of these experiments. Other investigators [6] have stated that the diuresis which may be caused by alcohol, or the bulk of fluid taken with it, leads to frequent urination so that the extreme discrepancies between the concentrations in the blood and urine, such as those shown here, do not, as a rule, occur. No doubt such is generally true; under the conditions of ordinary medicolegal tests there is usually fair agreement between the concentrations of alcohol found in blood. But there are occasional exceptions, and it is with these that we deal particularly. We find that there are wide variations in the diuresis from alcohol; there is also wide variation in the volume of fluid taken with the alcohol. Two of the eleven subjects employed here showed no diuresis with the amount of alcohol given them. In the remaining nine, diuresis occurred. The diuresis started in the second half hour after the ingestion of the alcohol and persisted for one and one-half hours.

The average volume of urine secreted for the full eleven hours of the experiment was 930 cc.; the individual extremes were 484 and 1,298 cc. During the first two hours, the time of the diuresis, the average amount of urine secreted was 390 cc.; the individual extremes were 91 and 612 cc. The subjects who had the greatest diuresis suffered considerable discomfort in retaining the urine during the first four or six hours, columns 8 and 9; those who had little or no diuresis had no desire to urinate. None of the subjects suffered discomfort when urine was voided at two hours and again at eight hours, column 11. The average volume collected at the latter time was 296 cc.; the individual extremes were 208 and 396 cc. The concentration of alcohol in the blood at this time was 0.037 per cent but, as estimated from the urine, 0.121 per cent.

Concentrations of Alcohol in Blood and Urine

Time After Ingestion of Alcohol, Hours	Concentration of Alcohol in Blood, per Cent	Concentration of Alcohol in Blood as Estimated from Urine Voided at Times Indicated (Ratio of Solubility of Alcohol in Blood and Urine Taken as 1:1.3)								
½	0.152	0.087								
1	0.193	0.163	0.141	.....	.....	.....	.....	.....	0.149	
1½	0.191	0.192								
2	0.185	0.188	0.191	0.171	.....	0.176	.....	.....	.....	0.169
2½	0.171	0.177								
3	0.160	0.165	0.172							
3½	0.141	0.146								
4	0.129	0.133	0.143	0.171	0.166	.....	0.169			
4½	0.115	0.111	.....	.....	.....	.....	0.116			
5	0.103	0.109	0.121							
5½	0.091	0.100								
6	0.078	0.082	0.095	0.106	.....	.....	.....	0.158		
6½	0.066	0.070	.....	.....	.....	.....	.....	0.069		
7	0.057	0.062	0.067							
7½	0.049	0.051								
8	0.037	0.043	0.049	0.057	0.089	0.106	.....	.....	0.150	0.121
8½	0.030	0.033	.....	.....	.....	0.031	.....	.....	0.034	
9	0.023	0.026	0.032							
9½	0.016	0.021								
10	0.007	0.012	0.016	0.024	.....	.....	0.087			
10½	0.003	0.005	.....	.....	.....	.....	0.004	0.051		
11	0.000	0.003	0.006	.....	0.039	.....	.....	0.002		

The discrepancies which, as we have shown, may occur between the concentration of alcohol in the blood and in the urine can be avoided by making the test from the urine by the following procedure, which takes advantage of the close correlation of the concentration in blood and in urine taken at half hour intervals. The suspect is requested to empty his bladder; the specimen is discarded or, if the concentration of alcohol is determined, it is taken as having only qualitative significance or it is used in conjunction with the subsequent determination to judge whether the concentration in the blood is rising or falling. One-half hour later the individual is again requested to void. Even the small amount of urine collected is sufficient for the determination of the concentration of alcohol. The value obtained is

6. Bavis, D. F., and Arnholt, M. F.: The Specificity of Methods for Determining the Concentration of Ethyl Alcohol in Body Fluids with Specific Reference to the Heise Method, Nebraska M. J. **32**: 407 (Nov.) 1938.

divided by 1.3 and the dividend is taken as the concentration of alcohol in the blood at the time the test is made.

The concentration thus found will lie between the concentrations in the blood at the beginning and at the end of the half hour period. If the concentration of alcohol in the blood is falling, the concentration as found from the urine will not be higher than that which existed in the blood at the time the suspect was taken into custody and requested to void. It will be lower than this value by less than the small amount that the concentration had fallen during the half hour period. If the concentration in the blood is still rising from the absorption of alcohol, the concentration as found from the urine will again fall between the two concentrations in the blood but will be higher than the first and lower than the second. Values obtained in this manner have definite quantitative significance which can be defended in court.

An occasional individual may be unable to void within a half hour. If necessary, a somewhat longer time may be given without altering the principle that the concentration as found is that which existed within the time between the two voidings. The probable extent of the error introduced can be judged from the values given in the table.

RELATION BETWEEN THE CONCENTRATION OF ALCOHOL IN BLOOD AND IN URINE

In the kidney, alcohol passes from the blood into the urine by diffusion;[3] the amount of alcohol in the two fluids is determined by the respective solubilities of alcohol. For urine collected over short intervals the ratio of solubility (with blood taken as 1) has been determined by Southgate and Carter[7] as 1.37, by Carlson, Kleitman, Muehlberger, McLean, Gulicksen and Carlson[8] as 1.32, by Jetter[9] as 1.23 and by Bavis[10] as 1.22. In the present study we have obtained a value of 1.31 when urine was collected at half hour intervals. We have in addition determined in vitro[5] this ratio for urine of different specific gravity; at 1.028 the ratio was 1.27, at 1.012 it was 1.30 and at 1.002 it was 1.34. The slight differences in solubility resulting from alteration in concentration of the urine, or as found by different investigators, are of little importance since the extremes do not vary more than 5 or 6 per cent from an average value of 1.30. These variations are well within the limits of accuracy of most of the clinical methods used for the analytic determination of alcohol in urine.

ABSORPTION OF ALCOHOL FROM THE BLADDER

The question of absorption of alcohol from the urine into the blood has been raised with regard to the use of the concentration in the urine as an index of that in the blood.[11] Völtz, Baudrexel and Dietrich[12] in 1912 showed that when alcohol is injected in considerable quantities into the bladder of a dog it disappears from the urine and appears in the blood. This work has been quoted as indicating that the rate of absorption is nearly independent of the concentration.[13] In reality Nicloux and Nowicka[14] in 1913 demonstrated that it varies with the concentration. They found that, when the concentration in the urine was brought to 1.5 per cent, 25.8 per cent of the alcohol was lost in two hours and that, at 2.2 per cent, 43.6 per cent was lost in this time. The concentrations used by these investigators were far in excess of the differences in concentrations in blood and urine found in medicolegal tests. We do not doubt that alcohol may be absorbed from the bladder but, in the low concentrations appearing after alcohol is drunk, the absorption is insignificant.

The possibility of absorption was, however, tested experimentally on men by two procedures. Two of the subjects employed in the present study gave no diuresis from the amount of alcohol given. When they had taken no fluid after 10 o'clock at night, their hourly secretion of urine during the following morning was virtually uniform and was not affected by the drinking of 180 cc. of water containing 40 Gm. of alcohol. On alternate days, to a total of ten, the urine was voided at half hour intervals so that no appreciable difference in the concentrations of alcohol in the urine and in the blood could develop. On the remaining five days the urine was held in the bladder for eight hours to obtain a maximum difference between the concentrations in the urine and in the blood. For one subject the urinary output, when voided at half hour intervals, was 424 cc.; the concentration of alcohol was 0.049 per cent. When the urine was collected for the entire period the volume was 437 cc. and the concentration 0.046 per cent. The corresponding values for the other subjects were 480 and 505 cc. and 0.054 and 0.051 per cent. The close correspondence of the concentrations under the two conditions would indicate that at least after moderate amounts of alcohol there is no appreciable absorption from the bladder.

In the second series of experiments, no alcohol was taken by mouth. The blood and urine were tested to prove the absence of alcohol. Urine was allowed to collect in the bladder for three or four hours; a catheter was then inserted and held in place. Under sterile conditions the urine was withdrawn and measured. Sufficient alcohol was added to give a concentration between 0.22 to 0.41 per cent and a sample was analyzed to determine the exact concentration. The amount of alcohol in the bladder was calculated from the volume and percentage. The urine was then run back into the bladder and allowed to remain there for two hours. The bladder was then emptied and washed twice with 60 cc. of water; the total volume of the fluid obtained was measured and the concentration of alcohol determined. The amount of alcohol recovered was calculated from these values. For one subject 492 mg. of alcohol was introduced into the bladder; 466 mg. was recovered two hours later; the loss was 5 per cent. For the second subject 223 mg. was introduced and 207 mg. was received two hours later; the loss was 7 per cent. In these experiments all error is in the direction of failure to recover alcohol, since appreciable amounts may remain in the film of moisture left on the surface of graduates, catheter and bladder wall. The slight losses of alcohol found here indicate that, at concentrations in the urine found on medicolegal examination, absorption is of no practical significance.

7. Southgate, H. W., and Carter, G.: Excretion of Alcohol in the Urine as a Guide to Alcoholic Intoxication, Brit. M. J. **1**: 463 (March 13) 1926.

8. Carlson, A. J.; Kleitman, N.; Muehlberger, C. W.; McLean, F. C.; Gulicksen, H., and Carlson, R. B.: Studies on the Possible Intoxicating Action of 3.2 per Cent Beer, Chicago, University of Chicago Press, 1934.

9. Jetter, W. W.: Studies on Alcohol, Am. J. M. Sc. **196**: 475 (Oct.) 1938.

10. Bavis, D. F., and Arnholt, M. F.: Test of Blood and Urine of Drunken Drivers, Nebraska M. J. **24**: 220 (June) 1939.

11. Miles, W. R.: The Comparative Concentrations of Alcohol in Human Blood and Urine at Intervals After Ingestion, J. Pharmacol. & Exper. Therap. **20**: 265 (Nov.) 1922.

12. Völtz, W.; Baudrexel, A., and Dietrich, W.: Ueber die Resorption des Alkohols durch die Harnblase, Arch. f. d. ges. Physiol. **142**: 186, 1912.

13. Carpenter, T. M.: The Metabolism of Alcohol, Quart. J. Studies on Alcohol **1**: 201, 1940.

14. Nicloux, M., and Nowicka, V.: Contribution a l'étude de la perméabilité et du pouvoir absorbant de la vessie, J. de Physiol. et de path. gén. **15**: 297, 1913.

CONCLUSIONS

In the medicolegal diagnosis of intoxication the concentration of alcohol in the blood is frequently estimated indirectly from that found in the urine.

Unless special precautions are taken, serious errors may occasionally occur.

The ratio of the concentration of alcohol in blood to that of urine varies only slightly with the specific gravity of the urine; the ratio as determined here is 1:1.3.

Absorption of alcohol from the bladder into the blood does not occur to a significant extent at the concentration found in the urine after drinking alcohol.

THE TREATMENT OF FRACTURE OF THE EXTERNAL TIBIAL CONDYLE

(BUMPER FRACTURE)

JOSEPH S. BARR, M.D.
BOSTON

There is, even in recent literature, a wide diversity of opinion as to the best method of treatment of fracture of the upper end of the tibia. The problem can be simplified somewhat, I believe, if one fairly large group of cases is segregated and the treatment outlined in some detail. No attempt will be made to cover the whole field of fractures of the upper end of the tibia.

Of seventy-five consecutive cases of fracture of the upper end of the tibia admitted to the Massachusetts General Hospital Fracture Clinic, thirty (40 per cent) were fractures of the outer condyle without associated fracture of the fibula. This represents the largest single type of fracture in this region. It has been recognized as an extremely serious fracture by practically all observers, not because of nonunion, which rarely, if ever, occurs, but because it involves a weight bearing joint, and loss of normal knee joint function is the inherent danger.

TYPE OF INJURY

Three types of injury account for these fractures: (1) a direct blow on the outer aspect of the knee, (2) a fall from a height or (3) a twisting injury (tables 1 and 2). As the automobile bumper accounted for only 47 per cent of these cases, bumper fracture is not a particularly good name for it. The twisting injuries were all very similar, i. e. the victim's foot while walking or running caught in a hole, and the sudden twisting violence transmitted to the knee caused the injury.

PATHOLOGY

An understanding of the normal and pathologic anatomy involved is a prerequisite to correct treatment. The stability of the knee joint is not due to the depth and coaptation of the joint surfaces as is the hip but to the extremely strong supporting ligaments and surrounding musculature. The internal and external lateral ligaments in particular are extremely resistant structures. The normal inward inclination of the femoral shaft causes more stress to be placed on the outer than the inner femoral and tibial condyles.

If a person is attempting to elude an oncoming automobile but is not quick enough to escape, his leg will often be struck on its outer aspect at or just below the knee joint. The victim's foot fixed on the ground and the inertia of the body weight prevent displacement of the leg. There is, therefore, a force which, acting on the outer border of the knee, tends to produce a knock-knee deformity (genu valgus). The extremely strong internal lateral ligament and the inner muscular structures resist the tendency for the inner femoral and tibial condyles to separate. The outer femoral condyle, which transmits most of the body weight to the tibia, is therefore driven down into the central portion of the outer tibial condyle. The central portion of the articular cortex of the outer tibial condyle is usually comminuted and depressed below its normal level into the cancellous portion of the bone, and in addition there is a bursting outward of the outer margin of the articular cortex of the tibia with an associated longitudinal fracture running downward toward the shaft of the tibia (fig. 1). This outer fragment is almost always of fairly good size and is roughly triangular with the base of the triangle upward. It is to be particularly noted that this fragment does not displace downward, as it is held in place by the intact fibula, which acts as a buttress, and by the strong capsular ligaments attached to the margins of the joint. The external semilunar cartilage is usually torn sufficiently from its normal attachments to allow it to displace downward somewhat into the central cavity. Even in unreduced cases, however, it rarely, if ever, produces symptoms due to locking.

TABLE 1.—*Type of Injury*

	Cases	Per Cent
1. Direct blow on the outer aspect of the knee		
(*a*) Automobile bumper	14	47
(*b*) Other	5	17
2. Fall from a height	1	13
3. Twisting injury	10	33
Total	30	100

TABLE 2.—*Age and Sex*

Age			Sex	
Youngest	Oldest	Average	Male	Female
28	79	51	13	17

TREATMENT

The degree of displacement of the fractured fragments is the key to rational treatment.

A. *Minimal Displacement.*—In these cases (fifteen, or 50 per cent, of our series) there is by x-ray examination less than one-fourth inch displacement of the fragments, and an excellent anatomic result can be anticipated if the fracture is protected until consolidation is complete. The details of treatment can be varied within wide limits without jeopardizing the result. My method is immobilization in a carefully molded plaster cast or splint or in a Thomas splint with a Pierson attachment and with the knee in slight flexion until the swelling of the joint has subsided. This usually requires from one to two weeks. Aspiration of the joint effusion during the acute phase is permissible but is not necessary. Daily physical therapy is then instituted. At first this consists in baking, massage and quadriceps setting exercises. Gentle active motion of the knee always within the limits of discomfort is added to this program within another week or two. This is continued until normal musculature and a range of motion well beyond right angle flexion is obtained. The patient may be up on crutches after the first week or

From the Fracture Clinic, Massachusetts General Hospital.
Read before the joint meeting of the Section on Surgery, General and Abdominal, and the Section on Orthopedic Surgery at the Ninety-First Annual Session of the American Medical Association, New York, June 14, 1940.

two, but no weight bearing is permitted for a minimum of ten weeks after injury. A few intelligent and cooperative patients are allowed to begin guarded weight bearing with crutches and an elastic bandage applied to the knee, but the majority are fitted to a long caliper brace with a knock-knee strap which passes around the

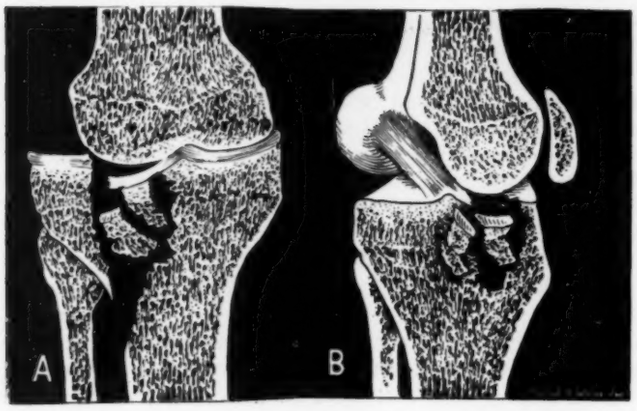

Fig. 1.—Saggital (*A*) and coronal (*B*) sections through the knee to show the usual type displacement of the fracture.

outer upright and pulls the knee toward it, preventing any tendency to occurrence of knock-knee deformity (fig. 2). The brace is worn for from two to six months and then discarded. If there is a tendency to pronation of the foot, a Thomas heel is used. The results of this type of treatment have been uniformly satisfactory in my experience.

B. *Slight to Moderate Displacement.*—Seven (23 per cent) of the cases were classified in this group, the displacement of the fractured fragments amounting to between one-fourth and one-half inch, and it is here that variance of opinion as to proper treatment is found. Three of these cases in the series were treated conservatively. One had a closed manipulation and three were subjected to open reduction. So far as I can ascertain from this small series of cases, the one year end results were about equally satisfactory. Whether a longer follow-up will reveal the development of instability, pain, hypertrophic degenerative joint changes and so on in more cases treated conservatively than operatively or vice versa remains to be seen.

C. *Moderate and Marked Displacement.*—Eight (27 per cent) of the cases in this series had displacement of fragments amounting to one-half inch or more, as estimated from x-ray appearance. It is my opinion that open reduction and fixation is the only satisfactory method of treatment in this group of cases. If unrecognized or deliberately left unreduced, the result is a painful, weak, unstable knee, which shows on examination marked abnormal lateral mobility. Increasing knock-knee deformity and marked hypertrophic changes occur as time elapses.

Manipulation cannot possibly effect anatomic reposition of the joint surface of the tibia. The comminuted depressed fragments in the central part of the tibial table are free fragments, have no ligamentous or muscular attachments, and lie in their depressed position surrounded by bone. So far as these fragments are concerned, manipulation is completely ineffectual. The outer larger laterally displaced fragment is not displaced downward and so does not need elevation. The use of a nutcracker vise or a carpenter's screw clamp has been recommended. They may effect reduction of the lateral displacement, but the central fragments are thereby impacted in their displaced position and the articular surface of the tibia remains distorted. Satisfactory anatomic reduction cannot be anticipated by this maneuver. It is axiomatic that fractures involving weight bearing joints require as nearly anatomic reduction as possible if good function is to be obtained.

A satisfactory method of open reduction must include (*a*) direct inspection of the joint surface so that the accuracy of the alinement of the articular cartilage may be verified, (*b*) an approach to the centrally displaced fragments from below so that they can be individually manipulated into place, (*c*) replacement of the laterally displaced outer fragment and (*d*) fixation of the fractured fragments so that early motion may be started and stiffness and atrophy avoided.

The technic to be described fulfils these requirements. It is not a new operation in the strict sense of the word, but it combines certain features of several previously described operations and is accepted by the members of our fracture clinic as the most satisfactory solution of the problem so far available.

TECHNIC

The operation is always performed under a tourniquet, as a bloodless field is essential. The skin incision begins about 1 inch lateral to the superior pole of the patella and extends downward just lateral to the tibial tubercle, then curving outward to end about 4 inches below the joint line just anterior to the fibula (fig. 3). The skin and subcutaneous tissues are reflected laterally off the muscles underlying the lower part of the incision until the head of the fibula comes into view. Attention is then turned to the joint, which is thoroughly inspected through a longitudinal incision in the capsule just lateral

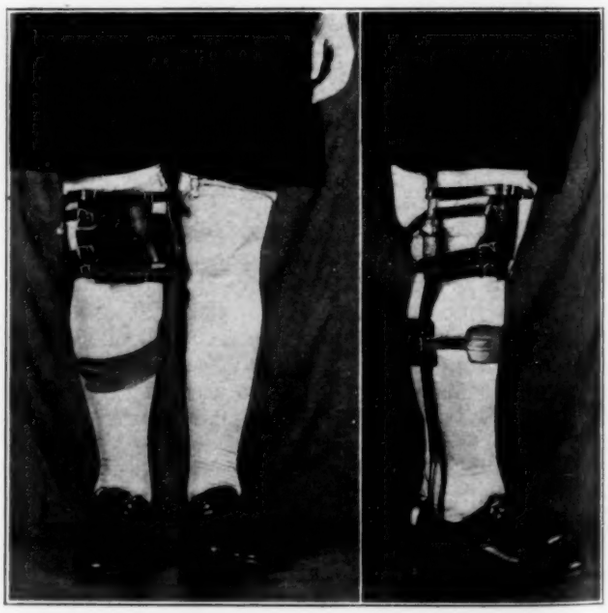

Fig. 2.—The brace has a knock-knee strap which prevents recurrence of the original deformity during the early stages of weight bearing.

to the patella. In order to visualize the extent of the fracture, it is usually necessary to remove the external semilunar cartilage, after which the whole articular surface of the outer condyle of the tibia is easily inspected.

Next the longitudinal fracture line is exposed by subperiosteal stripping (fig. 4) of the common origin of the extensor muscles from the anterolateral surface

of the outer tibial condyle. The incision is ⌐ shaped. The horizontal limb is from one-half to 1 inch long. Beginning at the tibial tubercle, it extends laterally parallel to and just below the anterior margin of the joint. The vertical limb extends downward for 2 or 3 inches from the tibial tubercle, just lateral to the crest of the tibia. The incision is carried through the periosteum and the muscles are reflected with the periosteal elevator laterally until the vertical fracture line is exposed. Care should be taken to leave the muscles attached to the lateral fragment, which is retracted laterally, opening the fracture line and giving access to the central part of the tibial condyle. The depressed fragments of articular cortex with cartilage attached can then be carefully replaced, by means of a blunt dissector or bone grasping forceps. In some instances, additional bone chips removed from the tibial shaft may be packed beneath the replaced fragments. The articular cartilage of the tibial condyle should present after this step a smooth anatomic restoration of normal contour. The operation must be considered a failure unless this is obtained. One then replaces the lateral fragment snugly, being sure that it locks the other fragments in jig-saw puzzle fashion. It can easily be demonstrated at operation that the replaced fragments tend to slip unless the large lateral fragment is firmly immobilized. Many methods have been tried, beef bone and ivory pegs, wood screws, autogenous bone grafts. None have proved as satisfactory as anchorage to the intact cortex of the medial tibial condyle by "bolting." This is mechanically secure; the compressive "bottling" effect of the bolt effectually prevents displacement and allows early motion to be begun. A Sherman screw, preferably of vitallium or stainless steel, with a washer over the head and a nut on the free end is used. It is sufficiently strong, and there is little or no tissue reaction about it. To introduce this bolt, a long drill of suitable size (No. 32) is inserted through the superficial lateral aspect of the lateral fragment and is directed slightly posteriorly and medially parallel to the joint surface to emerge through the medial tibial cortex just posterior to its subcutaneous surface. The drill point, felt through the skin, is brought out through a small skin incision. While the fragments are carefully held in place, the drill is removed and the Sherman screw with a washer over its head is inserted and tightened down snugly with the screw driver. A long enough screw is used (from 3 to 4 inches) so that the end projects well beyond the cortex on the medial side. The nut is then applied and tightened, so that there is no possibility of the screw loosening and losing its effective grip. The excess length of the screw is cut off with a heavy double action bolt cutter. With the knee joint under direct inspection, the knee is flexed to a right angle to check on the alinement and immobilization of the fragments. Unless there has been a technical error the knee is stable, there is no lateral mobility and the aim of the operation, anatomic restoration of the joint, has been accomplished. The wound is closed carefully in layers without drainage. The tourniquet is removed and a molded plaster splint and pressure dressing or a circular plaster in slight bow-leg position is applied (fig. 5).

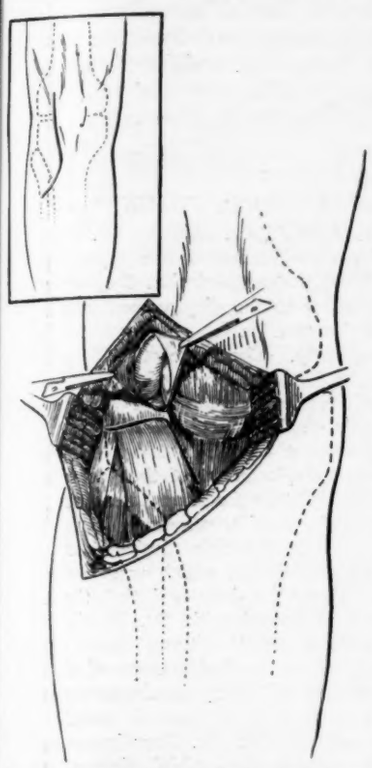

Fig. 3.—Operative approach. The knee joint has been opened for inspection, and the incision for the reflection of the muscles from the tibial condyle has been outlined.

POSTOPERATIVE CARE

After the stitches are removed and the postoperative reaction has subsided, the same program is carried out as was outlined for fractures with minimal displacement. Early motion is a feature of this schedule, and by the time weight bearing is permitted (from ten to twelve weeks after operation) there is usually an excellent range of motion present. Within six to twelve months after operation there should be a range of motion from full extension to right-angle flexion. The knee should be stable and an essentially painless normal gait should be present. It may not be necessary to remove the screw unless it shows signs of bone absorption or irritation around it, but it has been my practice to remove all "hardware" about one year after its introduction.

RECONSTRUCTION OF THE MALUNITED TIBIAL CONDYLE

Although the restoration of a condyle which has united in a deformed position is more of an orthopedic than a fracture problem, a word on it may not be amiss. In one case I have secured an excellent result by the following procedure: The operative exposure was identical with that used for fresh fractures, the joint being opened through a longitudinal incision and the outer condyle exposed by stripping the muscles from it subperiosteally. A window of cortical bone was then removed from the anterior aspect of the lateral condyle of the tibia just below the joint line. A small thin osteotome inserted through this window was used to make multiple stellate fracture lines in the displaced articular cortex of the tibia, care being taken not to injure the articular cartilage. It was then possible to

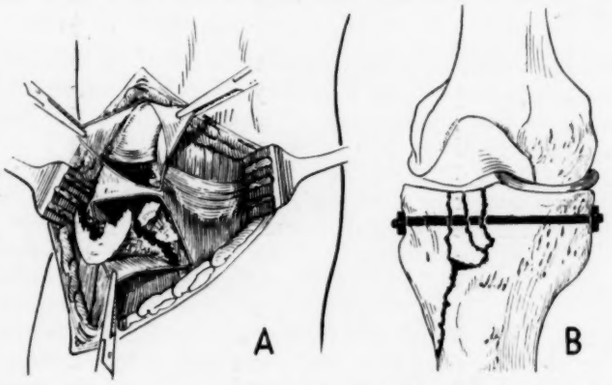

Fig. 4.—*A*, lateral bony fragment still attached to the muscles and the loose small fragments in the center of the condyle. *B*, screw in place.

force the joint cartilage and articular cortex upward into normal position by means of slow molding pressure with a blunt instrument. The bony cavity below was packed with bone chips. Although the widening of the tibial condyle is unchanged by this procedure, the central part of the tibial table is restored to normal level.

END RESULTS

It is pertinent to point out that various members of the fracture clinic have cared for these patients and that treatment in certain instances has varied from the theoretical "ideal" program that has been outlined. It is against the background of constantly increasing clinical experience that the present ideas of treatment have been formulated.

The results in group 1 (minimal displacement) have been uniformly good, no matter what treatment was adopted.

In group 2 (moderate displacement) satisfactory functional results were obtained in the three operative cases and in the four cases treated conservatively, but examination of the end result x-ray films reveals, of course, in the latter group the same degree of displacement present originally. A follow-up of at least five to ten years will be necessary to determine whether conservative or operative treatment offers the best solution to these borderline cases.

Fig. 5.—*A*, preoperative fracture. Note the two fragments displaced downward as well as the laterally displaced one. *B*, after operation. *C*, end results. The knee is stable and painless and has a full range of motion.

In group 3 there were five cases treated by the operative technic described. These plus 3 cases from group 2 make too small a number for statistical verification of the adequacy of the operation. However, seven of the eight cases showed an excellent anatomic restoration, and the functional results were equally good. The period of disability averaged eight months. In the remaining case there was only a fair anatomic and functional result, apparently because of faulty operative technic.

SUMMARY AND CONCLUSIONS

Fracture of the outer tibial condyle without associated fracture of the fibula accounted for 40 per cent of all fractures of the upper tibia in this series and is the most common single type encountered. For purposes of treatment it is convenient to classify them with regard to the amount of displacement, i. e. slight, moderate or marked displacement of the fractured fragments. This fracture assumes a fairly constant form in which there is comminuted depression of the central portion of the articular surface of the lateral condyle, and a large marginal fragment is displacel laterally. Manipulation is futile. The severely displaced fracture should be treated by open reduction and fixation, and an operative technic has been developed which has proved satisfactory in my hands. Although it is technically rather difficult and should be undertaken only by those experienced in surgery of the knee joint, it seems to afford the best anatomic and functional result. The after-care of the operative case and of the conservatively treated mild fracture is identical.

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ABSTRACT OF DISCUSSION

DR. CARL E. BADGLEY, Ann Arbor, Mich.: Dr. Barr has emphasized the need of a classification of the types of fracture that occur to the tibial plateau and that they should be analyzed and treated according to the displacement and to the indications. The type of fracture he is speaking about is one that is seen the most frequently. There are other fractures of the tibial plateau which he has not discussed. This fracture will occur to the knees in various positions of flexion and a greater amount of posterior displacement will frequently occur without the lateral displacement of the lateral tibial plateau. There is a great downward displacement of the posterior half or two thirds of the tibial plateau in some instances, which also requires the operative form of treatment. I have found in cases in which there was considerable displacement of the tibial plateau with associated injuries which were sufficiently severe that operation could not be done and that the use of Russell traction, which is in accord with Dr. Barr's method of support, has given excellent results without operative treatment. I feel, however, that with displacement of the joint fragments and the possibilities of injury to the meniscus and of nonunion of the fracture there is a group in which operation is indicated. I do not feel that Dr. Barr has completed the classification for the treatment of tibial plateau, but he has given us a good start so that ultimately we shall have more or less definitely outlined in mind the type of cases that can be treated by conservative methods.

DR. EARL D. MCBRIDE, Oklahoma City: I agree with Dr. Barr that the two major objectives in treatment are (1) to restore the knee joint architecture and (2) to reduce and maintain replacement of fragments. In my experience the x-ray examination does not disclose the full extent of the pathologic condition in most cases. I have found the cartilaginous surface of rather large fragments turned completely upside down, yet the x-ray examination showed very little displacement or fragmentation. This is due, of course, to the lack of density of bone in this region. The operation which the author describes for open reduction is a procedure of choice. He is cautious about recommending open operations except in the third class of cases with marked displacement. I so often find more pathologic change than was expected that I would recommend exploration, at least in many of the cases of apparently mild displacement. On the other hand, I have been surprised at the good functional result in several cases in which the reduction was very unsatisfactory from the x-ray standpoint. My experience is similar to that of Dr. Barr in that the shorter screws and nails are unsatisfactory. If long screws are used and placed in a converging manner, contrary to the tension of displacement, they often hold satisfactorily. In several cases I have treated this type of injury by fixed traction, placing the patient on a Hawley table with a wire above and below the region of fracture and forcing the leg in the direction of correction of the deformity and putting it up in fixed plaster in that position. I have used magnesium pegs in two or three of these cases when there was a rather small fragment with moderate displacement but when I wanted it to hold. A small peg placed in there will hold and gradually absorb. A bone peg from the area below on the tibia may be used. That forms a nice method, but unfortunately one has to make a larger incision, with more trauma about the region of the fracture, which I do not like.

DR. JOSEPH S. BARR, Boston: My point in presenting this paper was to isolate a certain type of fracture of the upper end of the tibia and to discuss the treatment of that particular group of fractures and no other. Fractures of both tibial condyles, comminuted fractures and fractures involving the fibula with downward displacement do not fall into the group that is being discussed. For that reason I still feel that I can safely say that traction and manipulation have no place in the treatment of this particular group of fractures, that the displaced outer condyle cannot be changed in position by either manipulation or traction, that if one is going to do anything at all for it one must do it by open reduction. In those fractures with minimal displacement, the external semilunar cartilage remaining in place has the effect of being a buffer between the mildly displaced fragments and the femoral condyle, and a smooth articular surface will reform if early motion is started and the surfaces are protected until union has occurred. In cases of moderate displacement there is certainly room for argument. I think perhaps one might be a little more radical and explore those with moderate displacement, but the immediate results are apparently about as satisfactory with conservative as with open operation. In the third group with marked displacement I think there can be no room for argument that the fragments must be reduced. The question is how they shall be held in place. I have tried various methods and have failed in maintaining reduction with all methods, with the exception of this one, which fixes the fragments firmly by securely holding the displaced fragments to the intact medial cortex. This is not an operation to be undertaken lightly. It requires major surgery to the knee joint and must be done by those experienced in knee joint surgery. If the operation is undertaken and one fails to secure a smooth anatomic reposition of the fragments, one has failed in the operation and the patient is worse off than he was at the start.

ANOXIA

THE ANESTHETIST'S POINT OF VIEW

RALPH M. WATERS, M.D.
MADISON, WIS.

Need for the present day reviving interest in nosology is illustrated by the terminology used in this symposium. By derivation, the meaning of anoxia is "without oxygen," a condition incompatible with life and therefore scarcely a suitable word to use in clinical discussions. For want of such a word as "hypo-oxia" or "hypoxia," anesthetists are in the habit of using the clumsy expressions "oxygen want," "oxygen lack" and "oxygen deficit."

Illness, injury and the exigencies of surgery cause pain and often an associated oxygen want. Pain relieving drugs have side effects which interfere with the mechanism of respiration. Hence if suffering is to be safely abolished or even minimized, the prevention and treatment of oxygen want must go hand in hand with drug administration. The delivery of oxygen from the respired atmosphere to cells of the central nervous system may be thought of as a transport mechanism. A simple diagram is frequently used in teaching the dangers of faulty oxygen transport and means of avoiding them. The student is advised to transpose his diagnosis of the condition of the patient before, during and after anesthesia into concepts applicable to such a diagram. In a consideration of the mother and fetus in utero, the diagrammatic concept is extended to include the mother's pelvic blood supply, the placenta, the umbilical cord and the circulatory system of the fetus. Into such a concept it is easy to interpolate diagnostic observations and the indicated therapeutic procedures.

The circumstances which embarrass oxygen transport with which anesthesiology is concerned may be classified in four groups:

(*a*) The condition of the patient before anesthesia.

(*b*) The pharmacologic characteristics of drugs other than pain relief.

(*c*) Disturbances of physiology due to technical difficulties of drug administration.

(*d*) The contributions of surgery.

THE CONDITION OF THE PATIENT

Because of his illness or injury, the oxygen transport mechanism of the surgical patient is frequently defective. Consciously or unconsciously, the anesthetist must evaluate every patient's physical status in terms of ability to deliver oxygen to the central nervous system. The normal patient tolerates temporary oxygen deficit surprisingly well. Preexisting defect in transport reduces tolerance in geometric proportion to the gravity of the defect. The history and the physical and laboratory examinations are searched for indications of defective oxygen delivery. Is there evidence of hypersensitive autonomic reflexes or attacks of syncope? Has there been bleeding? What is the quantity of hemoglobin, the number of red blood cells, the functional capacity of the heart, the pulse pressure, the condition of the arteries? Is the spleen normal? Is there evidence of or a history of pneumonia, pulmonary tuberculosis or fibrosis? So important does the anesthetist consider patency of the air passages that he rarely trusts the preoperative examination of the upper respiratory tract to another. A preanesthetic record of temperature, pulse and respiratory rate and blood pressure is important as a control with which to compare observations made during and after anesthesia. Administration of anesthetics for so-called emergency operations to patients without preliminary examination has led to frequent morbidity and mortality due to oxygen lack which, if anticipated, might have been prevented. Insistence on preoperative hospitalization for from twenty-four to forty-eight hours has saved many lives.

PHARMACOLOGY

Sedatives and narcotics administered orally or hypodermically as well as anesthetic agents which are injected or inhaled for the prevention or relief of pain have similar effects on the mechanism of respiration. Almost without exception they depress the respiratory center, decreasing minute-volume exchange, the extent varying with the dose of the drug and the susceptibility of the patient. Obstruction to ventilation likewise is an indirect result of many pharmacologic effects. Secretions of the mucosa and salivary glands are either increased or decreased and made more viscid. The pharyngeal, laryngeal and vomiting reflexes may be made hyperactive or obtunded and their normal coordination distorted. The cough reflex and ciliary activity may be paralyzed, thus depriving the respiratory tract of its normal janitor service. The muscle relaxation, so beneficent for the patient's comfort as well as the surgeon's convenience, may if neglected result in disaster. A relaxed tongue, obstructing the glottis, has not infrequently initiated a fatal anoxia.

From the Department of Anesthesia, University of Wisconsin Medical School.

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TECHNICAL CONSIDERATIONS

Second to second supervision of the physiologic processes of anesthetized patients is essential for safety. Far more important is it than the choice of drug or even the dosage. The management of patients requiring pain relief after operation or in the medical wards is no less important although somewhat less exacting. A schedule of dosage and time of administration of morphine sulfate for postoperative pain relief is a disgrace to the hospital in which it is written. Individualization in the choice of agents and the doses in which they are given from minute to minute or from hour to hour, as the case may be, will minimize interference with oxygen transport.

When occasion demands, proper atmospheric mixtures must be offered the patient to insure sufficient tension of oxygen in contact with the alveolocapillary membrane. Artificial airways must be available and utilized promptly and with skill. The respiratory center subject to severe oxygen lack is not susceptible of stimulation until the defect is remedied. The availability of cylinders of compressed oxygen as a part of every anesthetic apparatus and as therapeutic equipment in every ward has benefited many patients and is saving lives daily.

Diagram for teaching dangers of faulty oxygen transport and means of avoiding them.

Like all good things, however, oxygen is capable of doing harm. Extremely high tensions of oxygen help to maintain saturation of the hemoglobin (a pink patient) in spite of obstructed or depressed respiration. Diagnosis may thus be neglected and appropriate treatment delayed or omitted. Atmospheres containing largely oxygen, nitrous oxide or other rapidly absorbable gases are sometimes administered to patients when their breathing is temporarily stimulated (at the end of inhalation anesthesia or while subjected to carbon dioxide hyperpnea). If depressed breathing ensues immediately afterward, inactive or obstructed areas of lung may become rapidly atelectatic. It is thus obvious that, if oxygen deficit is to be avoided, even more important than the proper choice of drug and the proper dosage of that drug is the constant intelligent supervision of the physiologic processes of the patient throughout the period of drug effect.

CONTRIBUTIONS OF SURGERY

The oxygen transport deficits attributable to surgery are considered by many to be secondary to blood loss and traumatic shock. The anesthetist, however, believes that the perfection of modern surgical technic minimizes these factors. Instead, it is apparent that anatomic distortions necessary for the performance of certain operations or the dressings essential following them are more frequently the causes of inadequate transport. The extreme Trendelenburg position, the "gallbladder lift," or the lateral position may initiate poor exchange, pulmonary atelectasis or even circulatory embarrassment. Plaster casts and tight binders necessary to protect areas recently operated on may add to the inadequate tidal exchange of patients treated for postoperative pain.

Evidence does not support the conclusion that either anesthetic agents or surgical trauma frequently produce a direct effect on the heart. Unrecognized and untreated defective oxygen transport may result in fatigue of both the myocardium and the respiratory musculature. Indirect effects operating through a hypersensitive autonomic nervous system admittedly do, on occasion, cause circulatory depression or even asystole.

COMMENT

The induction of anesthesia, whether by injection or by inhalation, stimulates the experienced anesthetist to watch carefully for the slightest evidence of oxygen lack. Hyperventilation is not considered a reliable sign of oxygen deficiency and in the presence of nonvolatile premedication and of profound dosage of anesthetic agents it seldom occurs. The absence of cyanosis likewise is not assurance of ample oxygen supply. If either hyperpnea or cyanosis is present, the cause must be sought and removed if possible. The following incomplete list of symptoms and signs has been found useful to the anesthetist in determining the possible presence of oxygen transport deficiency:

In the operating room:

Vomiting.
Dilatation of pupils.
Muscle twitchings and contractions.
Circulatory abnormalities: Disturbed pulse rate or rhythm; increasing or decreasing pulse pressure; skin pale, cold and wet, or blue; delayed capillary refilling.
Respiratory abnormalities: Phonation, obstruction, depression, altered rate and rhythm; asymmetrical chest movements; prolonged expiration; hyperpnea rarely.

In the ward:

Psychic changes: Anxiety; restlessness; fear of death; incoordination; uncooperativeness; delirium; unconsciousness.
Headache.
Nausea.
Air hunger.
Increasing temperature.
Circulatory abnormalities: Precordial or substernal pain; increasing pulse rate; syncope; shock.
Respiratory: Increasing rate; periodicity; obstruction; depression; asymmetrical chest movements; mediastinal shift.

When one or more of these signs and symptoms is noted during anesthesia, the addition of oxygen to the respired atmosphere is logical, since a preexistent

abnormally low tension may readily be the cause of the difficulty. As artificial atmospheres are so commonly used, a diagnostic test may conveniently be applied. An addition of oxygen to the inspired atmosphere should first be made as one or two inspirations of pure oxygen. If a short period of apnea follows, it is almost positive evidence that marked oxygen want previously existed. Thus the diagnosis of the condition is confirmed, but not the cause, nor is a higher tension of oxygen in the respired atmosphere necessarily the proper remedy. If the oxygen want has developed because of some form of obstruction to the air passages, to atelectasis, to depression of exchange or to a defect in circulatory transport, only temporary advantage will be gained through oxygen administration unless the transport embarrassment is relieved. Whenever a deficient oxygen supply is suspected, every effort must be made to diagnose the cause accurately and remove it if possible, before dependence is placed solely on high oxygen tensions in the inspired atmosphere. Breathing excess oxygen may mask the transport defect. The result may be a pink patient, it is true, but often one whose circulatory and respiratory muscles are subjected to unnecessary strain and whose biochemical balance is seriously disturbed.

The practice of administering as a matter of routine an oxygen atmosphere rather than an atmosphere of nitrogen and only sufficient oxygen has undoubtedly resulted in frequent untreated physiologic disturbance and unrecognized obstruction and depression of breathing.

After anesthesia and operation, the necessity of treating pain carries with it the same dangers of interference with normal respiration as those accompanying anesthesia. Muscular relaxation, inspissated bronchial secretions, depressed ciliary activity and absent or obtunded cough reflex rob the patient of his natural defense against obstruction to the air passages. Decreased minute-volume exchange results. Here psychic disturbances (uncooperativeness, delirium) and rising temperature, and pulse and respiratory rate are the suggestive evidences of oxygen want until proved otherwise. With the pulse rate as a guide, the richest oxygen atmosphere available should be administered. When maximum reduction in pulse rate has been attained, the tension of oxygen in the inspired atmosphere should be decreased gradually until the minimum flow of oxygen is determined which will maintain a minimum pulse rate. Again one should beware of masking the diagnosis of the underlying cause. Proper treatment may be delayed through the thoughtless continuation of oxygen therapy without search for the defect in the transport mechanism.

CONCLUSIONS

It is important to realize that disturbance of oxygen supply to the central nervous system is one of the most common deleterious effects of anesthesia.

The anesthetist does well to look on the physiologic mechanisms involved in the delivery of oxygen to the tissues of the body as a simple transport system.

Depression of and obstruction to respiratory exchange are common sequelae of anesthesia and pain therapy. Intelligent management, for example the use of artificial airways and manual or mechanical increase of tidal exchange to prevent disturbance of oxygen transport and to restore normal transport when possible, is a necessary corollary to drug administration.

Forewarned is forearmed. The integrity of the patient's oxygen transport mechanism should be investigated before pain relieving drugs are administered.

Oxygen therapy (high oxygen tension in the inspired atmosphere) is only one way of treating oxygen want in the tissues. Accurate diagnosis will often point the way to restoration of normal transport of oxygen, thereby eliminating the necessity for oxygen therapy. Adequate oxygen tension should be administered to avoid an oxygen deficit, but one should not be lulled into inaction so that one neglects diagnosis and any treatment which may restore a normal transport mechanism for distribution of oxygen to the cells of the body.

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ABSTRACT OF DISCUSSION

DR. F. A. D. ALEXANDER, Albany, N. Y.: The two points I should like to make are, first, that the signs of anoxia in the dog cannot be assumed to be quantitatively similar to those in the human being and, second, that the signs of oxygen want produced acutely in the normal human being are different quantitatively if not qualitatively from those produced in the sick patient, in the asphyxiated human being or in the anesthetized human being. My associates and I have observed the signs of acute anoxia in more than 1,600 instances in our clinic during the course of nitrogen inhalation treatments for schizophrenia. The first evidence of anoxia is an increase in respiratory rate and volume occurring simultaneously with an increase in pulse rate. The respiratory stimulation increases progressively, as does the tachycardia. The systolic blood pressure rises gradually and varies considerably in different individuals. The diastolic blood pressure may rise, fall sharply or remain unchanged. Cyanosis is not a prominent feature if the patency of the airway is maintained. The neuromuscular changes are motor restlessness, clonic contractions beginning as twitchings about the face and spreading rapidly to the extremities, tonic-tetanic spasms, and finally opisthotonos or torsion-extensor spasms. Consciousness is usually lost at about the point at which the pulse rate has reached 120. At the height of the anoxia, when the patient is breathing almost 100 per cent nitrogen, the arterial blood oxygen content is usually less than 2 volumes per cent. Obviously such a degree of anoxia can be safely maintained for only a short period. Normal physiologic functions are restored rapidly when oxygen is administered to terminate the treatment. The most striking feature of these treatments was the absence of the so-called crisis with a peripheral circulatory collapse and bradycardia which occur in anesthetized patients or in animals or in patients subjected to more prolonged periods of subacute anoxia.

DR. R. D. MCCLURE, Detroit: In the Henry Ford Hospital Dr. Hartman demonstrated clearly, in deaths from anoxia, the changes in the brain. In many years of surgery in New York, Baltimore and Detroit, I have seen deaths on the operating table which occurred during nitrous oxide anesthesia. Many such deaths in the literature have been attributed to apoplexy, as no autopsy was done. It is now known that these deaths in the majority of cases are due to anoxia and are preventable. Many cases of physical or mental impairment following anesthesia have been reported and many more have not been reported. Two patients whose histories were reported a year ago in the *Annals of Surgery* were left practically decerebrated. One of them died a year later and another one some months later. There is reason to believe that milder grades of anoxia occur occasionally during anesthesia plus preoperative sedation and result in permanent damages to the brain which are not detectable by ordinary methods of examination. A student at the University of Michigan was leading her class in that particular group. She came to Detroit and had an impacted molar extracted. She went alone to the dentist. When she reached home that afternoon her vision was blurred. She was later taken to an ophthalmologist and glasses were prescribed. In school her companions noticed no change in her, but the teachers

did. Her work began to fall off and before many months had passed this girl had failed completely in her school work. She returned home and returned to the same dentist with an impacted molar on the other side. He said "Don't let anybody give you gas again for you can't take it." She had become blue; artificial respiration had been given. It is this type of case that we are so conscious of now. When we were doing the arterial punctures with Dr. Hartman, the doctor's arrival with the syringe to take the blood from the patient's artery acted as a signal to the anesthetist to turn more oxygen into her mixture. This led to the development of the photoelectric cell oxyhemoglobinograph, which the anesthetists is unable to deceive.

DR. ERNST GELLHORN, Chicago: This paper prompts me to elaborate at least briefly something of my ideas concerning anoxia and asphyxia. All physicians know that when we clamp the trachea in an animal the blood pressure first rises and then falls and the animal fails. This classic physiologic experiment shows the pathologic effects of severe asphyxia. It is a different condition, however, when one studies anoxia with a very mild degree of carbon dioxide excess. When one studies changes in oxygen concentration in pneumonia one finds that in some cases the oxygen saturation is decreased and in later stages of the disease there is also an increased carbon dioxide tension. I am inclined to consider this condition an attempt of nature to rid itself of anoxia, because one does not have here an anoxia with acapnia but anoxia plus some moderate carbon dioxide excess which will potently stimulate circulation, respiration and the whole autonomic nervous system and thereby attempt to restore normal conditions.

DR. NICHOLSON J. EASTMAN, Baltimore: Our procedure at the hospital in the treatment of asphyxia neonatorum is something as follows: In the first place, warmth. These babies are shocked. They are kept warm and put in an apparatus in which there is heat, a Kreiselman apparatus; the same effect can be brought about by hot water bottles and blankets. Second, posture, declination of the head at an angle of about 30 degrees to facilitate drainage of mucus and fluid from the trachea. Third, aspiration of mucus from the trachea and the posterior pharynx, in order to make certain of an open airway, because obviously artificial inflation is impossible unless there is a clear airway. Finally, the introduction of oxygen into the pulmonary alveoli, or at least into the smaller bronchioles by one of several methods. In hospital practice, as I have said, we use the Kreiselman machine, which we have employed for several years with complete satisfaction. If that is lacking, it is my opinion that if one is experienced and doesn't blow too hard, mouth to mouth inflation is a satisfactory substitute for some of the machines such as the Kreiselman apparatus.

DR. RALPH M. WATERS, Madison, Wis.: In talking about the signs and symptoms which have come to be associated with oxygen want, I tried to eliminate the ones that we feel clinically have anything to do with carbon dioxide. Every anesthetist recognizes that every anesthetic which he gives has the two associated possibilities of high carbon dioxide and low oxygen, and it is very difficult to separate them. I anesthetized a woman who, under the effect of two different agents, had a complete cardiac standstill, asystole, with finger pressure on both eyeballs. I purposely carried her through various planes of anesthesia to respiratory arrest with ether, and only then did this excessive reaction of the oculocardiac reflex disappear. With the other agents I did not carry her to respiratiory arrest and the reflex hyperactivity persisted. She was tested later without any anesthetic agent or drug effect at all, in the ward, and she had perfectly normal oculocardiac and carotid sinus reflexes. We had a male patient who at ward examination had a hyperactive oculocardiac and carotid sinus reflex of an extreme sort in which we could get a record of complete heart standstill. That man was anesthetized later with ether anesthesia and had a perfectly normal reaction under anesthesia. He had morphine and scopolamine previously. I cite these two cases as the extremes of what we encounter clinically of drug effect on these autonomic mechanisms.

ABDOMINAL BLOCK WITH GENERAL ANESTHESIA, FOR UPPER ABDOMINAL SURGERY

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The choice of anesthetic agents and methods of their administration for surgical procedures within the upper part of the abdomen is a major problem. Three individuals must be considered—the patient, the surgeon and the anesthetist. The patient wants a method which will spare him any physical or mental suffering during the operation and which will give him the least amount of discomfort postoperatively. The surgeon desires that a condition be produced which will enable him to complete the operation expeditiously without causing undue trauma such as is necessitated when relaxation is inadequate. The anesthetist wishes to use agents and methods which will cause the least variation from the patient's normal physiologic processes and at the same time satisfy the surgeon's desire for relaxation. The anesthesia should not constitute a danger to life nor should it contribute toward the establishment of a high incidence of complications. To a great extent the choice of agents and methods hinges on the price one is willing to pay for the degree of relaxation that the surgeon with whom one is working requires.

When considering relaxation, it is well to remember certain fundamental facts. In the stage of full surgical anesthesia the motor centers in the spinal cord normally responsible for muscular tone are affected; their nervous discharges are lessened and tonus is diminished or abolished. Production of heat is decreased and circulation is depressed, particularly in the phase of venous return. The heart gradually fails for lack of adequate quantities of fluid against which to pump. Postoperative pulmonary complications occur as secondary sequelae owing to the fact that with both the intercostal muscles and the diaphragm atonic atelectasis is likely to develop.

Adequate relaxation is essential for the efficient performance of surgical procedures within the upper part of the abdomen. Extreme generalized relaxation maintained over a prolonged period is usually unnecessary and is frequently fraught with dangerous complications during and after operation. The necessity is for expeditious yet efficient progress in the course of the operation. The anesthetist is obligated to produce adequate relaxation when it is most needed for exploration, for the critical periods during the operation when precise manipulations are being undertaken and for the closure of the incision. It is also essential that unobstructed respiration be maintained with as little respiratory depression associated with the anesthesia as is feasible.

As in all other major surgical procedures, preoperative medication has an important bearing on the operative course. The patient should arrive in the operating room in a sufficiently drowsy state that recollection of the event will be hazy. At the same time, he should not receive so much medication that his vital functions

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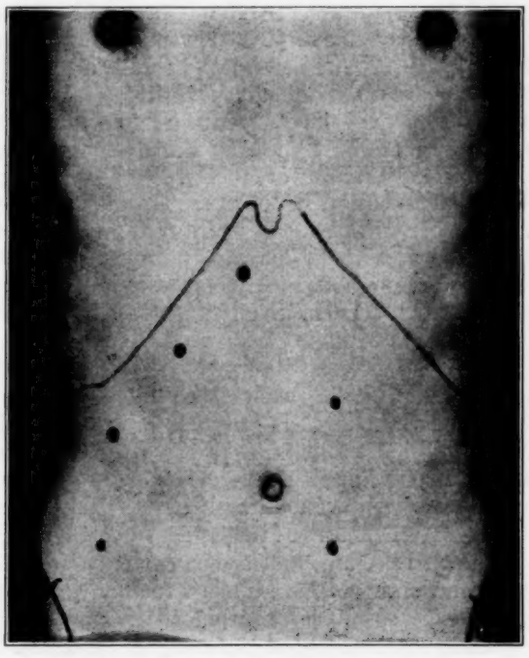
are inordinately depressed. The optimal state can usually be produced by administration of a moderately small dose of a barbiturate, such as pentobarbital sodium or sodium propyl-methyl-carbinyl allyl barbiturate given two hours before operation, and a moderately small dose of an opiate, such as morphine, given one hour before operation. If the patient is extremely apprehensive, pentothal sodium may be given intravenously while the patient is in his room. Some prefer administration rectally of a small dose of avertin with amylene hydrate in such instances. Atropine should be given to all patients, even when spinal or local anesthesia is contemplated. It serves not only to reduce the production of secretion within the respiratory tract but also to lower the irritability of the vagal centers.

There are several methods that may be used, either alone or in combination, to produce anesthesia for these operative procedures—inhalation, intravenous, regional and spinal. Inhalation anesthesia has been and still is widely used. Administration of the gases nitrous oxide, ethylene or cyclopropane may not result in the production of adequate relaxation unless ether is added. Pentothal sodium administered alone is inadequate for anesthesia for upper abdominal procedures. In combination with abdominal block with procaine, it has been used at the Mayo Clinic with good results. Pentothal sodium in a concentration of 2.5 or 5 per cent in distilled water may be used, and oxygen should be administered throughout the operation.

The use of regional anesthesia without the administration of supplementary agents to produce general anesthesia is restricted in most instances to patients classed as poor risks. Finsterer employs an abdominal block for the incision of the abdominal wall. He next inserts several long slender retractors and a minimal amount of packing, thus providing a narrow channel leading down to the region of the first lumbar vertebra. He then inserts a long trocar with a retractable needle attached and injects procaine in 0.5 per cent solution retroperitoneally. By this method, pain of visceral origin is controlled. For extensive gastric resections his mortality rate is only 4 per cent.

McCarty [1] describes a similar method and suggests that it may be the method of choice in certain types of cardiac decompensation, in acute respiratory and some chronic respiratory disorders, in oral or pharyngeal infections and in some cases of chronic nephritis and hypertension. He feels that it should also be used in the presence of marked or dangerous degrees of disturbance of acid-base equilibrium when time is not permitted to correct the condition. The method has never received wide acceptance in the United States, but Garth [2] suggests that many of the objections to the method could be obviated if general anesthesia produced by the intravenous administration of pentothal sodium should be employed during the manipulations necessary for the production of the splanchnic block. Lundy [3] has reported a series of cases in which abdominal block and splanchnic anesthesia produced though the posterior approach were employed. Adequate relaxation was achieved in only 50 per cent of the cases and the method was abandoned. Abdominal block, combined with the injection of from 75 to 100 cc. of 0.5 per cent procaine solution through the abdominal wall into the peritoneal cavity, has been employed with satisfactory results. The Trendelenburg position is employed in order to pool the free fluid in the region of the celiac ganglion.

The various agents and methods of their administration in spinal anesthesia for upper abdominal surgical procedures involve numerous and diversified factors which will be considered only briefly. In general it may be said that procaine will not produce relaxation of sufficient duration unless large doses are employed. The decrease in blood pressure will frequently be marked, and this reaction is frequently associated with the occurrence of nausea and vomiting. Pontocaine, although more toxic than procaine, may be used with the likelihood of sufficiently prolonged action. This drug may be dissolved in spinal fluid, mixed with procaine and diluted with spinal fluid or mixed with dextrose in 10 per cent solution. When the drug is dissolved in spinal fluid it is least controllable since it is practically isobaric. When it is mixed with procaine and diluted with spinal fluid the resultant mixture will in most instances be hyperbaric and the dose of pontocaine may be minimal. When it is mixed with dextrose 10 per cent a strongly hyperbaric solution will be obtained and sufficiently high spinal anesthesia may be most frequently attained, but the extension cephalad is so rapid that only an experienced anesthetist should employ the method. The latter method has been employed with considerable success by Sise [4] and his co-workers at the Lahey Clinic.



Points of injection for an abdominal block preliminary to a cholecystectomy.

Nupercaine is another agent used to produce prolonged high spinal anesthesia. It is used in a hypobaric solution in the manner outlined by the late Howard Jones. The drug is extremely toxic, and when it acts in untoward fashion cardiac action is influenced. It differs from both procaine and pontocaine in this

1. McCarty, F. B.: Infiltrative Anesthesia in Surgery of the Gallbladder, Am. J. Surg. **43**: 728-733 (March) 1939.
2. Garth, W. L.: Some Observations of Anesthesia Abroad, Anesth. & Analg. **17**: 292-297 (Sept.-Oct.) 1938.
3. Lundy, J. S.: Splanchnic Block, S. Clin. North America **5**: 887-893 (June) 1925; Balanced Anesthesia and Splanchnic Block, ibid. **6**: 1385-1395 (Oct.) 1926.
4. Sise, L. F.: Pontocaine-Glucose Solution for Spinal Anesthesia, S. Clin. North America **15**: 1501-1511 (Dec.) 1935.

respect, and because there is little that one can do to rehabilitate cardiac action in comparison to what one can do to improve respiratory action nupercaine should probably be reserved for those patients for whom a particularly long operation is contemplated.

Dodd and Hunter [5] and Organe [6] have reported the use of nupercaine in spinal anesthesia in reduced doses combined with the administration of cyclopropane and nitrous oxide respectively. The advantages of these methods are quiet respiration, absence of nausea and vomiting during the operation and absence of psychic disturbances. In general, it may be said that minimal doses of any agent introduced intradurally should be employed. It is preferable to find it necessary to administer supplementary general anesthesia to ten patients rather than apply resuscitative measures to one.

At Hartford Hospital it has been our practice, for an increasingly large percentage of patients, to use abdominal block supplemented by general anesthesia produced by the administration of cyclopropane and oxygen. An endotracheal tube is inserted whenever it is deemed necessary for the maintenance of an unobstructed airway. When surgical procedures involving the stomach are undertaken, the intratracheal tube is surmounted by an inflatable cuff. Protection against aspiration of material forced up the esophagus can be provided. Small amounts of ether are occasionally added to facilitate intubation or in those instances in which sufficient depth of anesthesia with cyclopropane cannot be maintained without having untoward symptoms develop. The method of carbon dioxide absorption is employed, thus permitting control of the character of respirations without the necessity of producing extremely profound anesthesia which is associated with partial paralysis of the respiratory center and extreme muscular relaxation. It is felt that it is preferable to produce quiet breathing by removing the natural stimulus, carbon dioxide, rather than by paralyzing the respiratory center by means of an excessive concentration of the anesthetic agent. With this method it is possible to produce apnea during the critical stages of the operation when active diaphragmatic movement would otherwise hamper progress of the procedure.

For abdominal block, a solution of procaine and corbasil in physiologic solution of sodium chloride is used. Originally we used a solution of procaine in 0.5 per cent concentration, but experience has shown that better relaxation can more uniformly be obtained by using a solution containing procaine 1 : 100 and corbasil 1 : 40,000. The patient is anesthetized with cyclopropane and oxygen or with pentothal sodium administered intravenously. The method of blocking the intercostal nerves supplying the abdominal muscles is a modification of one described by Lundy. No attempt is made to raise a wheal intracutaneously nor is any attempt made to infiltrate subcutaneously. A 50 mm. needle attached to a 10 cc. syringe is inserted through the skin. Resistance will be encountered as the needle approaches the superficial fascia. The fascia is pierced and, after initial aspiration to test against the possibility of intravascular injection, 10 cc. of the solution of procaine is injected. The needle is withdrawn and the procedure is repeated at intervals of 80 mm. along the costal margin and flank of the side of which incision is to be made, as shown in the illustration. Ten cc. of solution is deposited subfascially at each point through which the needle is introduced. Two injections into the upper part of the opposite rectus muscle completes the block. In patients with marked abdominal distention, in obese persons and in those who have a markedly distended gallbladder, great care must be taken not to introduce the needle too deeply. The peritoneum may be pierced and, although no damage will be done, the solution will to a great extent be wasted.

Reports of 300 patients undergoing 310 surgical procedures have been reviewed. Of the patients, 205 were women and ninety-five men. The youngest patient was 20 years of age and the oldest was 88. There were twenty-one patients between 20 and 30 years of age, sixty between 31 and 40 years, seventy-four between 41 and 50 years, seventy-three between 51 and 60 years, fifty-nine between 61 and 70 years and thirteen were over 70. For 139 operations the patients had inhalation anesthesia combined with abdominal block. This represents about 45 per cent of the series, and the predominance of this method was even greater in the latter part of the series. In seventy-nine instances endotracheal anesthesia was employed. The remaining 104 patients, 34 per cent of the group, received endotracheal inhalation. Fifty-one, or 16 per cent of the group, received inhalation only, and spinal anesthesia was employed for sixteen, or 5 per cent of the group.

An attempt has been made to evaluate the relative merits of the four anesthestic methods. In our opinion optimal conditions are produced with the greatest ease when abdominal block combined with the administration of cyclopropane and oxygen is employed. With this method relaxation could be obtained more frequently without the addition of ether vapor to the mixture, and when ether was employed the amount required was less than when the abdominal block had been omitted. The average operating time for patients receiving abdominal block was materially less than with the other methods that were employed. The incidence of postoperative complications was definitely less among the group receiving abdominal block than among those anesthetized by the other methods. Of the thirty-seven patients who died, six were subjected to secondary surgical procedures. For the forty-three operations, abdominal block was employed in only twelve instances.

SUMMARY

Abdominal block, as we have described it, is a relatively simple procedure. In hospitals in which the organization permits it, the block may be done by the anesthetist. Where this is not possible, the block can easily and rapidly be done by the surgeon before the incision is made. The method has been employed to advantage by us and it is becoming increasingly popular among surgeons. Satisfactory working conditions for the surgeon can be obtained without markedly disturbing respiratory function. It seems reasonable to limit marked muscular relaxation to the area for which it is necessary. This method is definitely preferable to infiltration of the muscles after the incision has been made. Tissues in the operative area are not edematous, and closure of the peritoneum is facilitated. A study of the incidence of postoperative complications indicates that abdominal block, employed in conjunction with inhalation anesthesia, is advantageous.

5. Dodd, Harold, and Hunter, J. T.: Cyclopropane "Sleep" with Nupercaine Spinal Anesthesia in Major Abdominal Operations, Lancet **1**: 685-688 (March 25) 1939.

6. Organe, Geoffrey: An Anesthetic Technic for Upper Abdominal Surgery, Anesth. & Analg. **18**: 339-342 (Nov.-Dec.) 1939.

ABSTRACT OF DISCUSSION

DR. JOHN S. LUNDY, Rochester, Minn.: It is important that methods of anesthesia be safe. There are several methods of local anesthesia used throughout the country. Infiltration is used, and abdominal block as you have heard it previously described is used when the skin is infiltrated as well as the subcutaneous tissues, and then Bartlett has just this year suggested blocking of the lower six intercostal nerves in the midaxillary line, which is very effective. I think that this combination of inhalation anesthesia and abdominal block offers a great deal without demanding any great penalty. Many times a method of anesthesia may be very valuable in a large number of cases and every now and again there will be one that will not do well under that method. I think the hazards associated with this combination are less than with most other methods. In regard to the intravenous method of anesthesia, I might suggest that it is possible to use 50 per cent of nitrous oxide and 50 per cent of oxygen simultaneously with intravenous anesthesia. The 50 per cent of oxygen is ample for the oxygen needs, the 50 per cent of nitrous oxide reduces the dose of pentothal sodium necessary, and I believe that you will like it.

DR. ROBERT L. PATTERSON, Pittsburgh: Satisfactory anesthesia from the standpoint of the surgeon, anesthetist and patient for upper abdominal surgery continues to be a problem in many institutions throughout the country. It does not hold true that deep anesthesia produces, for the surgeon, satisfactory working conditions for upper abdominal surgery. Accompanying the so-called deep anesthesia is encountered partial or complete intercostal paralysis resulting in maximum diaphragmatic movement to compensate for lost intercostal action. Maximum diaphragmatic movement increases greatly the movement of the stomach and intestine. This in itself makes for anything but a quiet abdomen. As we have seen from the paper of Drs. Tovell and Hinds, this situation can easily be overcome by using a deep abdominal block to produce the required muscular relaxation and still maintain intercostal activity. With the loss of general muscular tone there is also an associated loss of pharyngeal muscular tone producing varying degrees of respiratory obstruction. Similarly, high concentrations of the anesthetic agent in the mixture often produce laryngospasm, further increasing the degree of obstruction. Combating this obstruction further increases the work of the diaphragm. This too can be overcome through the use of an endotracheal tube, thereby assuring a completely patent airway throughout the entire operative procedure. The only objection to this method of anesthesia for upper abdominal surgery is the time consumed in doing the block. The usual time required is about five minutes and may be started immediately following the induction of anesthesia. The reduction in operating time resulting from thorough relaxation and the ease with which exposure is obtained more than offset the time consumed in doing the block. The benefits derived from this method of anesthesia warrant its continued and increasing use not only for fair and poor risk patients but for all patients subjected to upper abdominal procedures.

DR. RALPH M. TOVELL, Hartford, Conn.: I consider this method technically easier, more freely adaptable and probably safer than intercostal block. So far as surface anesthesia is concerned, I see little necessity for that. One has already induced general anesthesia with cyclopropane. The method is applicable by either anesthetists or surgeons. It seemed worth reporting because it is simple to accomplish and the results are satisfactory.

Celebrated Victims of Gout.—Many celebrated men in history have suffered from gout, notably Martin Luther, Samuel Johnson, Darwin, Newton, Milton, Gibbon, the great physician Sydenham, Lorenzo the Magnificent, Louis XIV, Louis XV, Louis XVI—men famous for their abnormal appetites and overindulgence in sweet wines. The familial tendency toward gout, so commonly supposed to be the only etiological factor in the disease, is probably much less important than faulty food habits. —Wolberg, Lewis R.: The Psychology of Eating, New York, Robert M. McBride & Co., 1936.

EARLY DIAGNOSIS AND PROGNOSIS OF GASTRIC CARCINOMA

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CHICAGO

Cancer of the stomach is the most important malignant tumor. Deaths from this disease constitute from one fourth to one third of all deaths due to malignant tumors. Approximately 4 per cent of all adults die from cancer of the stomach.

Gastric cancer is not a disease of senility, although it occurs especially after the thirty-fifth year. And that is the reason why the systematic fight against this frequent disease looms as one of the most important tasks of the medical profession.

The attitude concerning the prognosis of this fight seems to have changed. Physicians of the older generation were thoroughly pessimistic, and usually still are pessimistic. Although many of them have seen an occasional cure by early surgery, they have observed that the vast majority of all their patients suffering from gastric carcinoma died. Public opinion was likewise pessimistic. Too many cases were known in which surgery had been attempted but in which death occurred nevertheless either soon after the operation or one or two years later. This thoroughly pessimistic attitude was perhaps justifiable at the time in spite of the fact that in quite a few cases prolongation of life was procured by gastric surgery. In recent times, however, and especially in the minds of our younger physicians, a more optimistic attitude has arisen. This is due to the statistics on surgery of gastric carcinoma published by leading clinics. Now the students of medicine are told that 20 per cent of all patients in whom a resection of gastric cancer has been carried out will survive longer than five years.[1] And this certainly is an impressive statement. The mental attitude resulting from this optimism is a gratifying one. More patients are now examined by special methods, such as x-rays and gastroscopy, at a very early stage; and laparotomy is advised in almost every case which seems to offer a possibility of cure. Nevertheless, one still finds practitioners who wait much too long before carrying out x-ray and gastroscopic examinations of patients suffering from minor abdominal distress, slight lack of appetite and beginning

Figure 1.

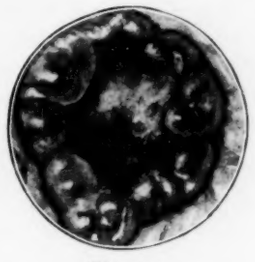

Figure 2.

Fig. 1.—Gastric carcinoma type 1. Gastroscopic view of antrum and pylorus. The curved line is the "angulus"; in the background the dark hole of the pylorus is seen; between 5 and 7 o'clock the fold of the "musculus sphincter antri" separates the antrum above from the body below. A polypoid nodular tumor, sharply circumscribed, is visible in the right side of the field. At gastroscopic examination the surrounding mucosa was completely atrophic and the tumor was dark red.

Fig. 2.—Gastric carcinoma type 2. Gastroscopic view. A large ulcer is seen in the center, surrounded by a nodular wall which is sharply limited toward the surrounding mucosa. In this view the nodular wall was dark red, the surrounding mucosa pale pink.

From the Department of Medicine, University of Chicago.

1. Livingston, E. M., and Pack, G. T.: End Results in the Treatment of Gastric Cancer, New York, Paul B. Hoeber, Inc., 1939.

loss of weight. Under such circumstances delay is not merely unjustifiable; it is almost inexcusable.

However, from a therapeutic standpoint one cannot be very optimistic. The statement that 20 per cent of patients obtain at least a five year cure refers only to those in whom resection is possible and ignores those in whom it cannot be carried out. Probably not more than 3 per cent of all patients with gastric carcinoma admitted to a clinic or hospital will be cured for five years or longer. And this is not a very encouraging result. However, these 3 per cent must be saved by all means, and therefore thorough examination is necessary and early surgery indispensable in every case in which the presence of metastases or the general condition of the patient does not make a successful operation impossible. This is not a satisfactory state of things. Many unsatisfactory laparotomies will in the long run destroy the confidence of public opinion and of the physician as well; therefore, one of the chief problems of the physician is to find out whether he can distinguish before surgical intervention between the types of carcinoma giving a good surgical prognosis and those giving a hopeless one.

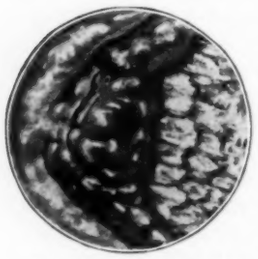

Fig. 3.—Carcinoma type 2, demonstrating the difference between the gross sharp limitation (fig. 4) and the microscopically undifferentiated character of the tumor (fig. 5). Gastroscopic view of the upper half of the tumor. At the right side the necrotic floor of the carcinomatous ulcer is seen. It is surrounded by a high sharply limited wall.

In a recent discussion on gastric carcinoma a pathologist, Dr. Paul Steiner, stated, in my opinion quite correctly, that our knowledge of gastric carcinoma is rather inadequate. We are, he said, at about the same stage as we were with brain tumors before some neuropathologists and neurosurgeons, especially Bailey, began to classify the brain tumors and to try to treat these tumors according to the classification. It seems to me that this would be the proper procedure in gastric carcinoma also. Now the physician does not differentiate between the prognosis of different forms of gastric carcinoma; he treats all of them in the same way. In my opinion, this is one of the chief reasons why there are so many failures in gastric surgery. If it could be established that gastric carcinoma resembles brain tumor as far as some forms give in the average a very good prognosis and other forms a definitely bad prognosis, then one could try to diagnose these different forms before surgical intervention. And the surgeon refusing to operate on forms with a bad prognosis would suddenly increase the percentage of successful operations. As a result, it would become known generally that if surgery for gastric carcinoma is undertaken the patient will usually be saved.

In the case of the brain tumor previously mentioned such a differentiation has been possible. Whether it will be possible in the case of gastric carcinoma also remains to be seen. Closest cooperation among practitioners, surgeons, roentgenologists and pathologists over a long period of years and a most careful work-up of every case, and especially of the pathologic material obtained, will be necessary. Steiner stated that it is not even known as yet which gastric carcinomas develop from the surface epithelium, which from the chief cells, which from the parietal cells, which from the Brunner glands and so on. Do all these tumors give similar pathologic or clinical pictures? As soon as the question of classification comes up (or may I say of typology, although this is not an accepted word) I feel that the study of gastric carcinoma is just beginning. Our present knowledge is not very extensive. It is known that gastric carcinoma has usually been subdivided according to rather superficial morphologic criteria such as differentiation of the cells, amount of connective tissue present and so on (adenocarcinoma, colloid carcinoma, medullary carcinoma, scirrhus). Almost every author describing the microscopic picture of gastric carcinoma adds his own classification, and all these classifications have proved not to be of great value for the practical procedure of the surgeon, and especially not for the prognosis.

CLASSIFICATION OF GRADES AND TYPES

Broders [2] has tried to grade various malignant tumors according to the malignancy of the cells seen. For this grading he used figures of percentage. I believe that this method is not very exact in the case of gastric carcinoma, because one may often see in a portion of the tumor well differentiated, benign looking cells and in other portions large sheets of undifferentiated cells which appear to be very malignant. However, one may attempt to estimate the degree of malignancy of a gastric tumor according to the apparent differentiation of the carcinoma cells. A tumor with a marked differentiation of cells with formation of tubules which look almost like those of a benign tumor may be called a grade 1 carcinoma. If the tubules become more irregular, if their epithelium consists of several layers and if papillae are seen in their lumens, the tumor may be called a grade 2 carcinoma. If the carcinoma is still able to form some tubules but consists mostly of coherent sheets of cells, it might be called a grade 3 carcinoma. If no formation of adenomatous tissue is seen and if the cells are of various sizes, the tumor may be called a grade 4 carcinoma. Livingston and Pack seem to believe that such grading may permit one to a certain extent to make statements about the prognosis of a gastric carcinoma, but I doubt whether this is true. They contend that 60 per cent of patients having had a resection for a grade 1 or grade 2 carcinoma will survive the opera-

Fig. 4.—Carcinoma type 2. Gross appearance of lesion shown in figure 3 after resection. The sharply limited character of the lesion is well seen.

2. Broders, A. C.: Carcinoma Grading and Practical Application, Arch. Path. **2**: 376 (Sept.) 1926.

tion for more than five years, as compared with 20 per cent of all patients having had a resection. If this statement were true it still would not add much encouragement, because grade 1 and grade 2 carcinomas are rare.

I believe that such a statement is even misleading. For more than ten years I have had the impression that it is not the microscopic structure on which the final prognosis of a gastric carcinoma will depend. At one time I observed an exceptionally small pyloric tumor which grossly did not show any sharp demarcation and was an invading type of carcinoma. However, the pathologist who examined this tumor microscopically stated that it consisted of tubules of such regular structure that he was inclined to diagnose it benign tumor. This evidently was a typical grade 1 carcinoma. Nevertheless, mucosal metastases developed four months later and the patient died a short time after that from generalized carcinomatosis. From this time on I had the growing impression that the microscopic type of a gastric carcinoma is less decisive for the final prognosis than its macroscopic character. In the case just mentioned a beautifully differentiated tumor was growing infiltratively and the early operation was of no avail.

Borrmann [3] in his chapter on gastric tumors has given a macroscopic classification of gastric carcinoma which is now widely accepted and which is used everywhere in the gastroscopic description of gastric tumors. Borrmann is a pathologist and therefore is able to describe only the pictures he sees in postmortem specimens. The pathologist is not able to state whether the types he describes are entities or whether there are transitions between the different types; that is, whether one type can at a later stage develop into another type.

The first gross type of gastric carcinoma is the polypoid carcinoma (fig. 1). This is a sharply limited growth often looking like a mushroom with overhanging edge, its surface presenting numerous nodes and nodules which usually are of different sizes. Only at a very late stage will ulceration of this surface develop. The mucosa surrounding this type of tumor is often thoroughly atrophic. This type is not a frequent one. According to my own statistics [4] it is found in 2.9 per cent of all gastric carcinomas, if carcinoma of the cardia is omitted. In my experience this type grows slowly and, it seems, never changes into another type but remains polypoid and sharply limited. Microscopically most tumors of this group are well differentiated adenocarcinomas, and it seems that their radical operation at not too late a stage gives a very good prognosis. However, these tumors are not frequent, and further observations are necessary.

Type 2 is a very important form (fig. 2). It is found rather frequently, namely in 17.6 per cent of all cases. It is an ulcer surrounded by an elevated wall. This wall has a steep slope toward the surrounding mucosa and is sharply demarcated all around against the surrounding mucosa. The floor sometimes shows necrotic material of all colors—brown, purple, gray, dark red or white. Sometimes necrotic, gray white crystal-like excrescences can be seen floating in the current of air introduced during the examination. The wall may be smooth, or more frequently it is nodular and contains shallow erosions. Its dark red color then is in marked contrast to the pale color of the surrounding mucosa. Since the surgeon tries to operate as soon as possible on these tumors he rarely has the opportunity to watch gastroscopically their course over a long period of time, but sometimes the patient may refuse the immediate operation and then such an observation may become possible. Within only one week the thickness of the wall may double. However, I have never seen this sharply limited wall become diffusely infiltrative at a later stage. The number of such observations is naturally still too small to permit the definite statement that such a transition is never possible. The observation, nevertheless, is an amazing one because the microscopic structure of these carcinomas is not a uniform one. They often show little differentiation and look rather malignant microscopically (figs. 3, 4 and 5).

Fig. 5.—Carcinoma type 2. Microscopic section through the edge of the tumor shown in figures 3 and 4 with the adjacent atrophic gastric mucosa. The tumor is sharply demarcated but consists, nevertheless, of undifferentiated cells of an apparently highly malignant character.

The startling point about these type 2 carcinomas is that metastases develop at a relatively late stage and often lead to an extremely satisfactory end result, to cures of long duration if an operation can be performed at not too late a moment, whatever their microscopic structure may be. These are the well known tumors which may reach the size of a fist, which can well be palpated through the abdominal wall and which are definitely movable. This is the form of carcinoma which at x-ray examination frequently gives the so-called meniscus sign of Carman. Figure 6 gives the x-ray appearance of a typical type 2 carcinoma presenting the so-called meniscus sign. Although this type 2 carcinoma was located almost immediately below the cardia, total gastrectomy led to a five year cure.

In the type 3 carcinoma of Borrmann (fig. 7) an ulceration is found also. This ulceration has a wall, but

3. Borrmann, R., in Henke, F., and Lubarsch, O.: Handbuch der speziellen pathologischen Anatomie und Histologie, Berlin, Julius Springer, 1926, vol. 4, pt. 1, p. 865.

4. Schindler, Rudolf: Incidence of the Various Types of Gastric Disease as Revealed by Gastroscopic Study, Am. J. M. Sc. **197:** 509 (April) 1939.

the wall is found only at one side of the ulcer and does not slope as steeply as does the wall of the type 2 carcinoma. There is a more gradual transition, and the ulcer itself, although sharply limited at one side, blends diffusely at the other side, and there diffuse and progressive infiltration of the neighboring mucosa is seen.

Fig. 6.—Gastric carcinoma type 2. Typical meniscus sign of Carman. The barium fills the cavity of the ulceration. The white margin that surrounds this cavity and interrupts the course of the normal gastric rugae represents the carcinomatous wall.

This type 3 carcinoma occurs in 16.3 per cent of all cases. Here again the microscopic pictures are not typical at all.

The type 4 tumor of Borrmann is the diffuse infiltrating type, which unfortunately occurs in 63.2 per cent of the cases. No sharp limit will be found anywhere toward the normal gastric mucosa either with the eye or by palpation; sometimes the entire stomach may be infiltrated (fig. 8). In this case, in which the stiff nodular infiltration is seen, the entire stomach was found gastroscopically to be involved at the very first observation almost two years before the patient died. Within this infiltration, ulceration may develop—shallow ulcers or deep ones. They never show the sharp edge of the typical benign ulcer. These type 4 carcinomas seem to be not very favorable tumors. I have never seen a complete cure after the resection of such a tumor, but naturally the material is too limited to permit any definite conclusions.

In the extensive research which should be undertaken within the next decades concerning classification or typology of gastric carcinoma, both the gross and the microscopic structure should be studied carefully with the hope of coming to a surgical prognosis based either on the microscopic examination of gastroscopic biopsies or on the gross gastroscopic or roentgenologic appearance of the tumor, without exploratory laparotomy.

Thus far I have been able to collect thirty-nine cases in which such a study was possible. The results are given in the accompanying table. The microscopic grades are listed, approximately estimated according to Broders' criteria; and the gross types are listed according to Borrmann. There is no microscopic grade 1 in this material. The four cases which would constitute a microscopic grade 2 are evenly distributed over the four types. One of these Broders grade 2 tumors belonged to the Borrmann type 1. The patient is still alive after two and one-half years and without any sign of recurrence roentgenologically and gastroscopically. Another Broders grade 2 carcinoma belonged gastroscopically to the Borrmann type 4; this patient, at the operation, exhibited carcinomatous invasion of all lymph nodes.

It is interesting to note that many of the sharply walled off type 2 carcinomas belong microscopically to grade 4. The fact that there is no relation between the microscopic grades and the macroscopic types is established, and this fact should be borne in mind for future research.

PRECANCEROUS CHANGES

So much for the classification and typology of gastric cancer. Now what can be said with regard to the subject of precancerous changes in the gastric mucosa? Today it is a widely accepted theory that carcinoma does not develop on the soil of an entirely healthy mucosa. Some European workers [5] go so far as to contend that no gastric carcinoma ever develops in a perfectly normal mucosa. It has been alleged that there are conditions of the mucosa which must be considered precancerous. If this is true, then evidently a new approach has been found. And this concerns the truly early diagnosis of gastric carcinoma. Every carcinoma starts with a few malignant cells. Perhaps one cannot hope ever to discover the stage in which only a few cells are malignant, but even in the most malignant growth there may be a stage of dividing malignant cells which have not yet invaded the lymph vessels but which are restricted to the gastric mucosa itself, forming a small pathologic area which may be recognized. Such truly early carcinomas, carcinomas of minimal size, cannot possibly cause any symptoms, even if they are located in the pyloric region. And therefore a truly early diagnosis of such carcinomas of minimal size can be made only incidentally. One should attempt to find them when

Comparison of Macroscopic Types and Microscopic Grades of Thirty-Nine Gastric Carcinomas

	Grades	1	2	3	4	Total
Types (of Borrmann)	I	..	1	..	..	1
	II	..	1	3	9	13
	III	..	1	3	4	8
	IV	..	1	3	13	17
	Total	0	4	9	26	39

Figure 7.

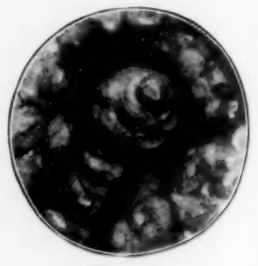

Figure 8.

Fig. 7.—Gastric carcinoma type 3. Gastroscopic view, showing along the greater curvature in the center of the field the wall of a small ulcerated carcinoma, but at the right side the carcinomatous infiltration reveals gradual transition into the gastric wall. Gastroscopically the gastric mucosa on the left side was gray and atrophic.

Fig. 8.—Gastric carcinoma type 4. Gastroscopic view showing stiff infiltration with formation of thick nodes.

5. Konjetzny, G. E.: Chronische Gastritis und Magenkrebs, Monatschr. f. Krebsbekämpf. **2**: 65 (March) 1934. Hurst, A. F.: Schorstein Lecture on Precursors of Carcinoma of the Stomach, Lancet **2**: 1023 (Nov. 16) 1929.

they do not cause any symptoms. If this could be accomplished then one might hope to discover all types of gastric carcinoma at such an early stage as to permit complete cure by immediate surgery. The way to accomplish this is to discover the precancerous conditions of the gastric mucosa and to watch such patients carefully. More important still is the task of finding the cause of these precancerous changes and a means of eradicating them before they develop further.

Fig. 9. — Adenomatous polyp of the stomach. The closing pylorus is seen in the center of this gastroscopic view and a cherry sized polyp is located in the pyloric ring.

What are these precancerous conditions? Three diseases have been considered forerunners of gastric carcinoma: (1) benign gastric ulcer, (2) benign gastric polyp and (3) chronic atrophic gastritis.

1. *Benign Gastric Ulcer.*—For a long time the opinion was prevalent that many gastric carcinomas develop on the soil of benign ulcers. This opinion, however, is now almost untenable. Dr. Walter Lincoln Palmer [6] contends that the existence of carcinomatous degeneration in benign ulcer remains to be proved conclusively. Gastro-enterologists who carefully watch their ulcer patients over a period of several decades have never seen such an ulcer develop into a carcinoma. Peptic ulceration of carcinoma may produce a lesion grossly indistinguishable from benign ulcer. Indeed, there is evidence that the carcinoma may be completely digested away by peptic activity, leaving a typical benign ulcer.[7] Probably a carcinomatous ulcer is malignant from the beginning. From 3 to 4 per cent of all adults die from gastric carcinoma, and therefore it has to be expected that from 3 to 4 per cent of all ulcer patients will develop a gastric cancer also; but the causal connection is hard to establish. For all these reasons it is no longer believed that benign ulcer is important as a precancerous condition. Ulcer patients who are being treated conservatively will naturally be watched, and development of a carcinoma in an ulcerous stomach will not be overlooked easily. Preventive resection of a benign ulcer to avoid development of carcinoma is hardly advisable.

2. *Benign Gastric Polyps.*—These are more frequent than is generally recognized. The smallest ones are easily overlooked at x-ray examination and, indeed, at postmortem examination. There is not yet a generally accepted classification of the benign gastric polyps. McGlone and I [8] have suggested that we consider three groups of them. The first of these groups belongs essentially to the entity of atrophic gastritis. The hemispherical excrescences, seen sometimes to cover the entire mucosa of the antrum, appear to be inflammatory formations growing on the soil of atrophic gastritis and consisting of connective tissue containing only a few tubules. However, Konjetzny has demonstrated that this type of pseudopolyps frequently develops into carcinoma and has recommended resection in such cases in order to prevent formation of carcinoma.

The second type of gastric polyp is the truly benign adenoma, being the size of a pea up to that of a cherry. If the polyp is the size of a cherry, as shown in figure 9, it may be well demonstrated by x-ray examination too (fig. 10). But if these polyps are very small they may escape easily the demonstration by x-rays. The gastroscopic picture is unmistakable. They may be single or multiple. These tumors are frequently found associated with atrophic gastritis also, especially in pernicious anemia; but the microscopic structure is that of a true adenoma. Their epithelium is undifferentiated, not showing any typical cells of the gastric mucosa. Grier Miller [9] and many other authors have contended that within these true adenomas gastric carcinoma will frequently develop.

The third type of benign mucosal polyp has been seen in only one case. This is the hyperplastic polyp originating in a hyperplastic mucosa. At operation in this case nothing could be felt at palpation from the outside of the stomach, but when the stomach was opened it was possible to remove with the fingers several rather hard benign tumors. Microscopically this type of tumor does not show the undifferentiated epithelium of the true adenoma, but it contains the cells of the gastric mucosa, chief cells and parietal cells; therefore, it can hardly be considered a true tumor but a kind of hyperplastic formation. I believe this should be called hyperplastic polyp. It is well known that in hyperplastic polyps of the intestine carcinoma frequently develops, but whether or not this is true for these hyperplastic polyps of the stomach is not yet known.

3. *Chronic Atrophic Gastritis.*—The recognition of gastric polyps is important, but from the standpoint of frequency and as a foreruner of gastric carcinoma this third condition to be considered is of much greater importance. Its clinical symptoms, the gastroscopic signs and the complications of this frequent disease have been described elsewhere. Pathologically it consists of a thinning of the gastric mucosa, the glandular apparatus of which disappears slowly and is replaced by cellular infiltration. The epithelium often undergoes transition into the intestinal type of epithelium with many goblet cells. At gastroscopy are seen thinning and graying of the mucosa with visible branching blood vessels.

Fig. 10.—Adenomatous polyp of the pylorus. X-ray relief compression view. In the upper half the cap of the duodenum is seen. In the lower half is the prepyloric region, which contains a sharply circumscribed filling defect.

Patients usually complain of minor epigastric distress, pressure, belching, bloating and sometimes ulcer-like pain. But their general symptoms—extreme fatigue coming in spells, headache, nervousness, numbness and tingling of the limbs—are more characteristic. If it is stated that this disease may frequently be the forerunner of gastric carcinoma, then this seems to be in complete contradiction to the old conception that gastric carcinoma usually develops in entirely healthy persons. But in the light of later investigations it seems that this old conception is true only in a

6. Palmer, W. L.: Benign and Malignant Gastric Ulcers, Ann. Int. Med. **13**: 317 (Aug.) 1939.
7. Ewing, James: Etiological Indications of Early Gastric Cancer, Rev. Gastroenterol. **7**: 305 (July-Aug.) 1940.
8. Schindler, Rudolf, and McGlone, F. B.: Familial Occurrence of Hyperplastic Gastric Polyps, Arch. Surg., to be published.
9. Miller, T. G.; Eliason, E. L., and Wright, V. W.: Carcinomatous Degeneration of Polyp of the Stomach, Arch. Int. Med. **46**: 841 (Nov.) 1930.

rather small percentage of cases. At different localities attempts have been made to clarify this question, and the respective workers have arrived at amazingly similar results. Kapp [10] of Basel, Switzerland, found that more than 13.4 per cent of his cases of gastritis later developed into carcinoma. The figure of Usland [11] of Drontheim, Norway, is 15 per cent, and this incidence is about three times that of gastric carcinoma in general. These observations support the observations of Saltzmann [12] and of Konjetzny [5] on atrophic gastritis leading to gastric carcinoma. I believe that one frequently does not study the case history of patients suffering from gastric carcinoma carefully enough. The patient seeing the physician at a rather late stage of the disease is overwhelmed by his experience of the last months, the disappearance of his appetite and the terrifying loss of weight, and he is inclined to forget that for many years he has been suffering from abdominal distress. On the other hand, I am not inclined to believe that every gastric carcinoma develops on the soil of atrophic gastritis.

If it is true that patients with atrophic gastritis have three times as much chance to develop a gastric carcinoma as healthy adults, then it is the obvious duty of physicians to diagnose this disease as early as possible and watch these patients closely by frequent x-ray and gastroscopic examinations. The correct diagnosis of atrophic gastritis is possible only gastroscopically, and the gastroscope in my opinion will have its chief significance in the diagnosis of this important disease. When the diagnosis is once established, routine rechecks should be made by x-ray examination and, if feasible, also by gastroscopy.[13]

Fig. 11.—Early diagnosis of an unusually small gastric carcinoma as a result of the routine examination of a patient suffering from atrophic gastritis. In the center of this gastroscopic view the distorted pylorus is seen containing a small grayish ulceration. At gastroscopic examination the mucosa of the antrum was greenish gray, indicating atrophic gastritis. Microscopic examination of the resected specimen revealed an early carcinoma 8 mm. wide and from 0.5 to 2 mm. deep.

Three cases which proved that such a procedure may lead to the discovery of extremely small carcinomas and to a really early diagnosis of gastric carcinoma have been published elsewhere [14] and will be summarized here briefly. In the first of these cases the early diagnosis was not made because of a misinterpretation of the x-ray film, but this case shows better than any other the typical course and the diagnostic possibilities that I have discussed:

A man aged 58 had suffered for twenty years from abdominal distress consisting of pains, belching, pressure and the like, all the symptoms coming on in attacks. Ten years before I saw him the gallbladder had been taken out and found filled with pus, but his distress continued. After this operation many tests of gastric acidity were made, always showing a histamine proved anacidity. There was a progressive state of weakness and fatigue developing into marked exhaustion on little effort. As a result the formerly active man was compelled to curtail his activities. Often he was forced to leave his work in order to have dietary treatment. This course, in my opinion, is one typical of atrophic gastritis. Severe attacks of weakness leading almost to incapacitation were present. Abdominal symptoms were found, and histamine proved anacidity was stated at repeated occasions. Three years before his admission to Billings Hospital a very small filling defect of the greater curvature was found and interpreted as a spasm. This apparent spasm was seen at several reexaminations. Retrospectively it is quite obvious that, by means of a gastroscopic examination and probably by a careful relief x-ray examination also, the presence of a tumor would have been diagnosed immediately. When this filling defect grew in size the patient wanted to have a gastroscopic examination (fig. 1). A type 1 carcinoma was found. This tumor was lying in a completely atrophic mucous membrane. Down from the cardia to the pylorus the gastric mucosa was thin and greenish gray, contrasting characteristically with the dark red of the tumor. The microscopic examination showed in the most perfect way the transition of a really atrophic mucosa into tumor tissue.

The second case does not show this connection quite so convincingly but it is striking because a minimum sized carcinoma was diccovered incidentally:

The patient had suffered from gnawing epigastric distress two hours after meals for one year, associated with belching and poor appetite. He had had several attacks of severe nausea. After his admission to Billings Hospital he had the usual complete check-up, and Dr. Frederic Templeton at x-ray examination was able to demonstrate a fleck suggestive of pyloric ulcer. The differential diagnosis between benign and malignant ulcer was difficult. The gastroscopic examination did not permit any doubt that this was a very small pyloric carcinoma (fig. 11). At gastric analysis 65 units of free acid was found; however, this frequently happens in atrophic gastritis. In spite of the definite gastroscopic diagnosis of malignant lesion, the surgeon was not able to feel anything definite at operation; but at our request he made a resection. In the gross specimen a very small, shallow ulcer was seen. It was assumed to be a benign ulcer, and only at microscopic examination was it shown that the floor of the ulcer consisted of carcinoma. This carcinoma had a width of 8 mm., and a depth of 0.5 mm. at one place and 2 mm. at another place. It is by far the smallest carcinoma that I have ever seen, and I am rather convinced that this small lesion did not produce symptoms of one year's duration but that these symptoms were due entirely to the atrophic gastritis.

In this case we were not able to prove definitely that the discovery of the small carcinoma was purely accidental, although this seems to be very likely; but in the following case there cannot be any doubt about the accidental discovery of a small carcinoma by a routine check-up:

A patient suffering from pernicious anemia and cord degeneration presented himself for a check-up. At x-ray examination nothing was found. Probably this was partly due to the fact that the patient had great difficulty in standing up because of his organic nervous condition. At gastroscopy the usual atrophic mucosa of pernicious anemia was seen, and when the gastroscope was rotated in the middle portion of the stomach a small type 3 carcinoma was seen to lie in atrophic mucosa (fig. 7). Microscopic examination revealed severe atrophic gastritis characteristic of pernicious anemia.

SUMMARY

The present approach to the fight against gastric carcinoma is not an entirely satisfying one. It has been suggested that a better classification of different forms be found in the hope that a reliable prognosis may become possible before surgical intervention. Emphasis has been given to the importance of the early diagnosis

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12. Saltzmann, Fredrik: Studien über Magenkrebs mit besonderer Berücksichtigung der Schleimhautveränderungen, Arb. a. d. path. Inst. d. Univ. Helsingfors, Jena: G. Fischer, 1913, p. 335.
13. Schindler, Rudolf: Die Bedeutung der Gastroskopie für die Heilung des Magenkrebses, Monatschr. f. Krebsbekämpf. **1**: 203 (May) 1933.
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and treatment not only of gastric cancer but also of the precancerous conditions of the gastric mucosa—polyps and especially the frequent and important chronic atrophic gastritis. Such early diagnoses are obviously possible only if routine x-ray and gastroscopic examinations are made. Only in this way will it be possible to discover true early carcinomas of minimal size and thus permit really early surgery and thus obtain a higher percentage of real cures and a greater prolongation of life.

TREATMENT OF FRACTURES OF ULNA WITH DISLOCATION OF HEAD OF RADIUS

(MONTEGGIA FRACTURE)

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Fracture of the ulna associated with dislocation of the head of the radius was described by Monteggia in 1814 and is often called by his name. During the past twelve years thirty-four articles dealing with this subject have appeared in the literature, and of these only two have been in English. The fact that the lesion is difficult to treat and the end results are often disappointing may account for the paucity of English literature on the subject.

The present study is based on a series of sixty-two injuries of this type which have been treated by the members of the staff of the Willis C. Campbell Clinic. Thirty of the patients had acute fractures, ten of which were compound. The remaining thirty-two patients had old injuries, having been first seen four weeks or longer after the accident.

The dislocation of the head of the radius was anterior in 83⅓ per cent of the patients, posterior in 10 per cent and lateral in 6⅔ per cent. The ulnar fractures were distributed as follows: 5 per cent involved the olecranon at the level of the elbow joint; 66⅔ per cent, including fractures at the junction of the upper and middle thirds of the ulna and excluding the 5 per cent involving the olecranon, were in the upper third of the ulna; 18⅓ per cent were in the middle third, and 5 per cent were in the lower third. In 5 per cent of the cases the dislocation was associated with a fracture of both the radius and the ulna.

MECHANISM

A Monteggia fracture may be produced by an indirect force, although as a rule the injury is caused by a direct blow on the forearm. Since the radius and ulna are well united by the interosseous membrane and the ligaments in the regions of the elbow and wrist, forces of this type ordinarily fracture both bones. Occasionally the annular ligament is ruptured and as a consequence the head of the radius is dislocated; or, even more rarely, the head of the radius is pulled out of the annular ligament. In either event the dislocation usually prevents fracture of the radius. If the force is excessive a dislocation of the radial head may be associated with a fracture of both bones of the forearm.

Read before the joint meeting of the Section on Surgery, General and Abdominal, and the Section on Orthopedic Surgery at the Ninety-First Annual Session of the American Medical Association, New York, June 14, 1940.

SYMPTOMS AND MANIFESTATIONS

The fracture of the ulna is usually evident from the history and physical manifestations. If the fracture is 2 inches or more below the elbow joint the proximal fragment is almost invariably angulated toward the radius, probably by the pull of the supinator muscle. The tendency toward this deformity is so strong that it will usually recur in a cast, even following open reduction and wiring of the fragments.

When one encounters a fracture of the ulna with angulation or overriding and without a fracture of the radius, a dislocation of the head of the radius should be suspected. In anterior dislocations the radial head can generally be palpated anterior and proximal to its normal location, this position being aided by the pull of the biceps tendon. A depression is palpable just anterior and distal to the capitellum. In posterior dislocations the head of the radius is palpable distal and posterior to the capitellum. In either type, attempts to pronate and supinate the forearm provoke pain at the elbow.

In making roentgenograms of fractures of the ulnar shaft, the elbow joint should always be included on the film. Failure to do so may lead to a grave error in diagnosis and to permanent deformity. One should not forget that, even in fractures of both bones of the forearm in the upper third, a dislocation of the radial head may be present.

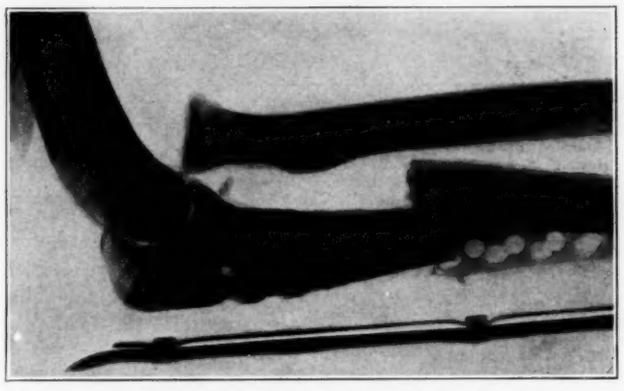

Fig. 1.—Lateral view of acute fracture of the ulna with dislocation of the head of the radius in patient aged 20 years.

TREATMENT OF ACUTE FRACTURES IN ADULTS

In some cases, manipulation will effectually replace the head of the radius and restore the fragments of the ulna to a satisfactory position. Following reduction of an anterior dislocation the extremity should be immobilized with the elbow in flexion and the forearm in supination; this position relaxes the biceps and supinator muscles, thus diminishing the upward pull of the biceps on the radial head and the radial pull on the ulna by the supinator. The position of flexion most successfully prevents recurrence of the dislocation. After correction of a posterior displacement, immobilization of the extremity with the elbow in extension and the forearm in supination may be necessary, since flexion of the elbow may lead to a recurrence of the dislocation; in this event, flexion should be gradually resumed as early as feasible. If possible, however, immobilization of the elbow in extension should be avoided. In only one of our cases was it necessary to maintain the elbow in extension. The patient obtained a good functional result, flexion and extension ranging from 45 to 180 degrees.

Seldom are good end results secured by closed reduction in adults. Replacement of the head of the radius is often difficult because of interference by the ruptured annular ligament, and, if the head of the radius is pulled out of the ligament, replacement by the closed

Fig. 2.—Lateral and anteroposterior views of same arm shown in figure 1 four months following fracture, showing radial angulation of the ulna and subluxation of the head of the radius. This illustrates the fact that wiring of the fragments will not prevent radial deviation of the ulnar fragment. Roentgenograms taken six years postoperatively showed a similar angulation of the ulna. The patient has severe limitation of pronation and supination.

method is practically impossible. Further, external fixation alone fails to prevent radial deviation of the proximal ulnar fragment. If the angulation of the ulna

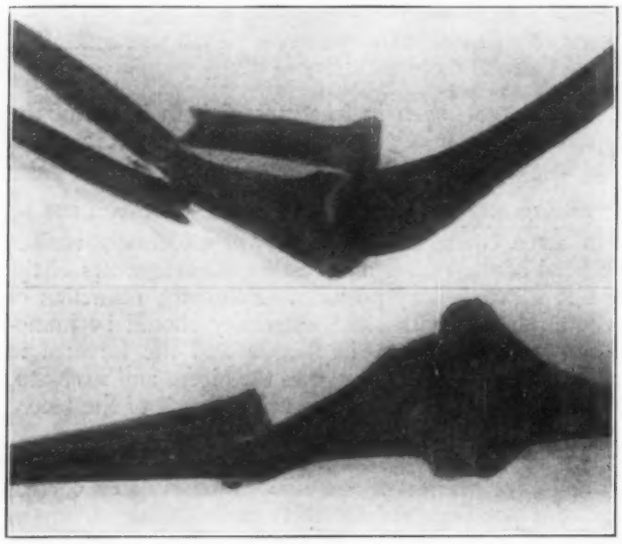

Fig. 3.—Lateral and anteroposterior views of acute fracture of both bones of the forearm with dislocation of the head of the radius in patient aged 17 years.

recurs sufficiently to produce an appreciable shortening, subluxation of the radial head will ensue or, if the fragments disengage and overlap, the dislocation will recur. The persistent tendency toward radial deviation of the ulnar fragment, usually associated with malunion and partial or complete recurrence of the radial dislocation, led to the treatment of these fractures by open reduction and internal fixation by means of a vitallium plate and screws. In our experience this method has proved the best insurance against angulation of the ulna, wire loops or other measures having failed to prevent radial deviation (figs. 1 and 2).

Reduction of the head of the radius may be maintained by (1) repairing the ruptured annular ligament or (2) passing a silk or wire suture or a strip of fascia around the neck of the radius and fixing it to the ulna. Both practically and theoretically the use of the fascial strip is the most satisfactory of these methods, since fascia forms a broad living ligament more nearly resembling the normal structure. Usually the annular ligament is too badly damaged to provide an adequate amount of tissue for a good repair, and silk and wire tend to cut into the neck of the radius and break following use.

When the head of the radius is fractured as well as dislocated, removal of the fragment or of the entire head is indicated, depending on the severity and extent of the fracture.

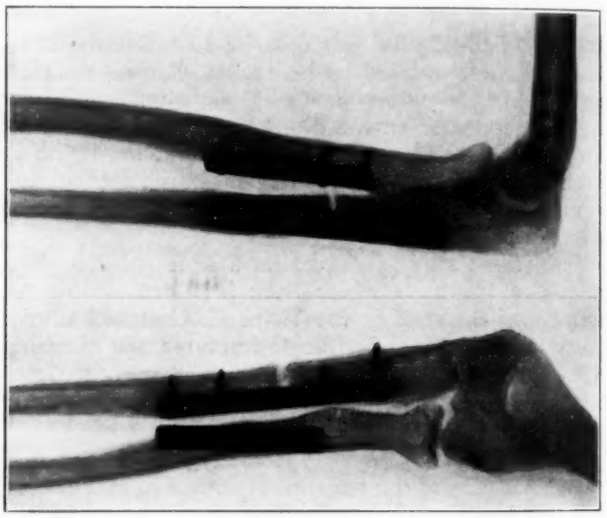

Fig. 4.—Lateral and anteroposterior views of same arm shown in figure 3 six months postoperatively. Vitallium plates were applied to the fractures of the ulna and radius, and a fascial loop was passed around the neck of the radius. The entire procedure was carried out through the one incision described in this paper.

The operation in its most elaborate form calls for an incision for exposure of the ulnar fracture, a second incision over the head of the radius and the removal of a strip of fascia lata from the thigh. In an effort to simplify the procedure, a single incision has been developed through which the fracture of the ulna and the dislocation of the head of the radius can be reached, and the fascia can be obtained locally to form a loop about the neck of the radius.

Operation.—An incision is made along the lateral side of the distal 1 inch of the triceps tendon and is continued distally along the subcutaneous border of the ulna as far as necessary for exposure of this bone. The insertion of the anconeus muscle is reflected subperiosteally from the upper 2½ inches of the ulna, together with the origin of the supinator muscle, that portion of the supinator which arises from the ulna distal to the radial notch being divided close to the bone. On reflection of this muscular flap laterally, the head and neck of the radius and the radiohumeral joint are exposed. The deep branch of the radial nerve is protected in the substance of the supinator muscle.

This approach extends only to the upper margin of the interosseous membrane. In the presence of a fracture of the upper third of the radius, reduction and internal fixation of the fragments are also possible through the one incision, by reflection of the muscles laterally along the dorsal surface of the membrane until the shaft of the bone is reached. In this manner, access is gained to the upper third of the radius without exposure or damage of the deep branch of the radial nerve. Separation of the muscles from the interosseous membrane necessitates division of the dorsal interosseous artery; no harm has resulted from ligation of the artery, however, in the two cases in which the more extensive approach was used (figs. 3 and 4).

In discussing open reduction in these injuries, Wilson [1] states: "Open reduction of the dislocation should be performed through an anterior incision after preliminary exposure and isolation of the radial nerve." Thompson [2] also advocates exposure of the upper third of the radius after isolation of the radial nerve. With this method one must incise the supinator muscle in order to isolate the nerve. In this region, moreover, the nerve gives off several branches, further complicating the procedure. Also direct exposure and traction on the nerve subject it to the possibility of injury. If

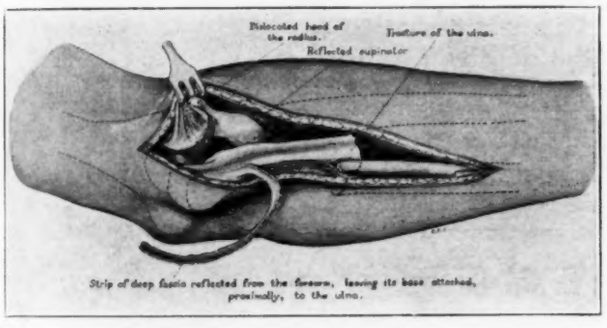
Dislocated head of the radius.
Fracture of the ulna.
Reflected supinator
Strip of deep fascia reflected from the forearm, leaving its base attached, proximally, to the ulna.

Fig. 5.—Drawing of incision described in the text. The anconeus and supinator muscles have been reflected from the ulna, revealing the head and neck of the radius. A strip of deep fascia has been elevated from the forearm and left attached proximally.

exposure of the upper third of the radius is required the method advocated herein is of particular value, since the supinator muscle can be elevated from the radius, thus providing ideal protection to the radial nerve as well as affording adequate access to the bone. This exposure has been described in detail in an article by one of us.[3]

After completion of the approach (fig. 5) the fracture is reduced and the position of the fragments maintained by means of a vitallium plate and screws. The head of the radius is replaced and any portion of the annular ligament which may be lodged in the radiohumeral articulation is removed. A strip of deep fascia, one-half inch wide and approximately 4½ inches long, is then elevated from the muscles of the forearm, being left attached to the proximal end of the ulna where the deep fascia blends with the periosteum at the lower end of the subcutaneous triangular space, which forms the dorsal portion of the olecranon. The fascial strip is passed between the radial notch of the ulna and the tuberosity of the radius and thence around the neck of the radius, and is fastened to itself with interrupted silk sutures. Thus, through this single incision the fracture of the ulna can be reduced and the dislocated head of the radius replaced and held in position by a fascial loop (figs. 6, 7 and 8).

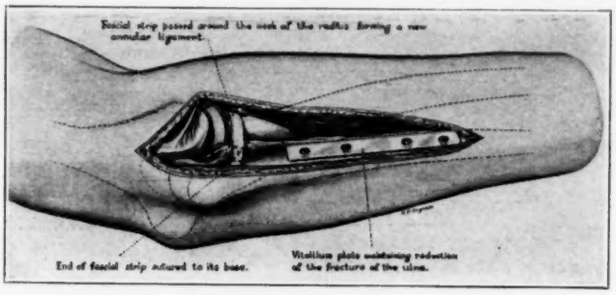
Fascial strip passed around the neck of the radius forming a new annular ligament.
End of fascial strip sutured to its base.
Vitallium plate maintaining reduction of the fracture of the ulna.

Fig. 6.—Operation completed. The fracture of the ulna has been reduced and a vitallium plate applied for fixation. The strip of fascia has been passed round the neck of the radius and sutured to itself, forming a sling for maintaining reduction of the head of the radius.

TREATMENT OF ACUTE FRACTURES IN CHILDREN

In children the fragments of the ulna can often be satisfactorily alined and the radial head replaced in the anatomic position by manipulation. In instances of anterior dislocation of the head of the radius the extremity is immobilized with the elbow flexed and the forearm supinated. In children, as in adults, the ulnar fragment deviates toward the radius; union takes place more rapidly, however, and as the child grows the angulation of the ulna tends to straighten out. An apparently severe malunion may be spontaneously corrected to an extent which permits normal function of the elbow

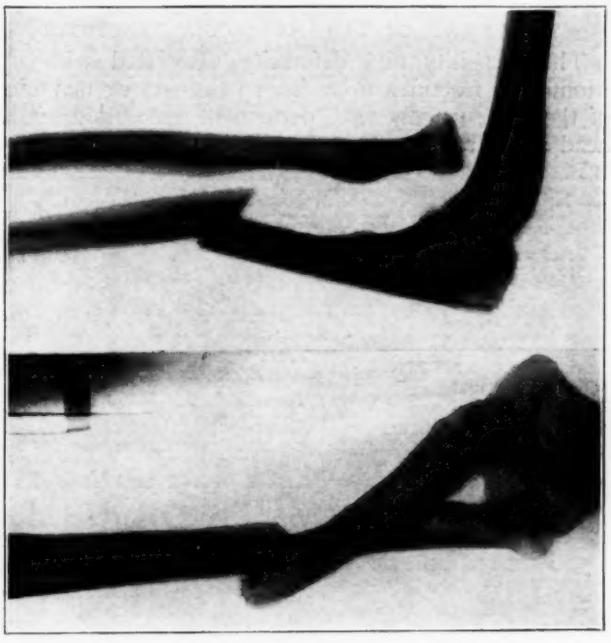

Fig. 7.—Lateral and anteroposterior views of acute fracture of the ulna with dislocation of the head of the radius in a patient aged 27.

(figs. 9, 10 and 11). Considering these beneficial growth factors, one is justified in accepting a more severe radial angulation of the ulnar fragment in children than in adults.

Maintenance of the radial head in the normal position is more important in children than anatomic alinement of the ulnar fragments. In the roentgenogram one may

1. Wilson, P. D.: Fractures and Dislocations in Region of Elbow, Surg., Gynec. & Obst. **56**: 335 (Feb., No. 2A) 1933.
2. Thompson, J. E.: Anatomical Methods of Approach in Operations on the Long Bones of the Extremities, Ann. Surg. **68**: 309 (Sept.) 1918.
3. Boyd, H. B.: Surgical Exposure of Ulna and Proximal Third of Radius Through One Incision, Surg., Gynec. & Obst. **71**: 86 (July) 1940.

find the head of the radius well reduced with the elbow in flexion, only to discover later, when the elbow is extended, that the dislocation recurs. In this event, open reduction of the dislocated radial head, combined with proper fixation to prevent recurrence, should be

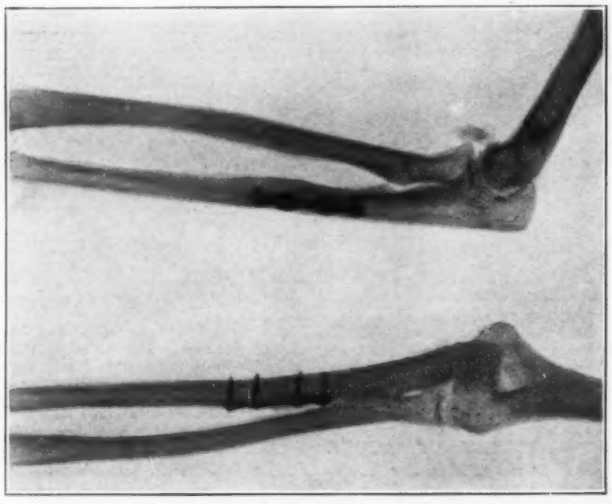

Fig. 8.—Lateral and anteroposterior views of same arm shown in figure 7. A vitallium plate was applied to the ulna and is shown, one year postoperatively, fixing the bone in excellent position in both views. The fascial loop that was placed about the neck of the radius has maintained reduction of the dislocation.

undertaken. Similarly, if one has difficulty in obtaining a reasonably anatomic alinement of the ulnar fragments, operation is advisable.

The surgical procedures described for the treatment of Monteggia fractures in adults are applicable also to those in children.

TREATMENT OF OLD MONTEGGIA FRACTURES

The disability and deformity associated with old Monteggia fractures arise from nonunion or malunion of the ulna, usually with permanent dislocation of the head of the radius. Occasionally one sees a permanent

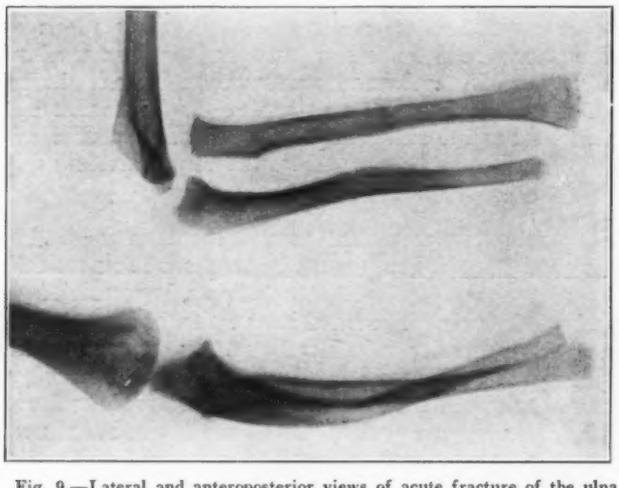

Fig. 9.—Lateral and anteroposterior views of acute fracture of the ulna with dislocation of the head of the radius in a patient aged 2½ years.

dislocation of the radius with only slight deformity of the ulna. In such cases the injury was generally sustained during childhood and the dislocation recurred or was never reduced.

Nonunion of the Ulna with Dislocation of the Radial Head.—In these old injuries the ends of the ulnar fragments are usually sclerotic, and in order to correct the angulation and secure bone of satisfactory quality for union one must resect the end of each fragment. This shortens the ulna from one-half to three-fourths inch. The muscles and other soft tissue structures of the forearm are contracted, incident to the angulation and shortening of the ulna. Under these conditions reduction of the head of the radius would produce a gap in the ulna of from one-half to three-fourths inch, which would be incompatible with bony union. Further, the pressure on the articular surface of the head of the radius would lead to degenerative changes. The outcome of such a procedure would be, in all probability, nonunion of the ulna, extensive arthritic changes in the radiohumeral joint or recurrence of the dislocation. Experience has shown that the safest procedure is resection of the head of the radius and a sufficient portion of the neck to compensate for the loss of length in the ulna, followed by approximation and fixation of the ulnar fragments by means of a bone graft (figs. 12 and 13). As an alternative, one might remove a sufficient portion of the shaft of the radius to compensate for the shortening

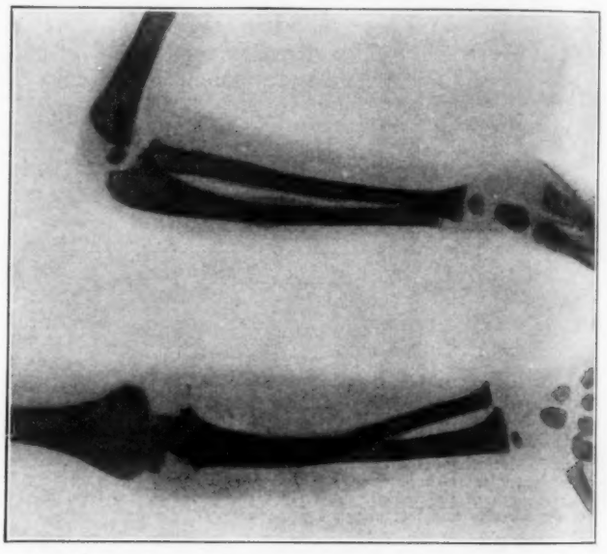

Fig. 10.—Lateral and anteroposterior views of same arm shown in figure 9 three weeks after reduction. The angulation of the ulna is still present; callus is present about the fracture site. The head of the radius remains in the corrected position.

of the ulna and apply a vitallium plate to the two fragments. There are, however, three objections to this procedure: (1) In many cases degenerative changes are present in the articular surface of the radial head which predispose traumatic arthritis in the radiohumeral joint, (2) the additional osteotomy of the radius increases the uncertainty of the outcome, and (3) the measure complicates and lengthens the operation. Moreover, the functional results probably would not be improved. This method was employed in one case of the present series in which a fracture of the radius was already present; although the fragments of the radius united, flexion and extension, as well as pronation and supination, of the elbow were severely limited from arthritic changes about the head of the radius.

Formerly, autogenous bone pegs were used for fixation of the onlay graft. In some cases, however, the pegs failed to maintain immobilization because of the strain at the fracture site produced by the radial angulation of the ulna. For this reason the pegs have been

discarded in favor of vitallium screws, which insure better fixation of the graft.

Malunion of the Ulna with Dislocation of the Head of the Radius.—Probably the safest method of treating malunion of the ulna with dislocation of the radial head is by osteotomy of the ulna at the point of maximum angulation and fixation of the fragments by means of a bone graft, as for the treatment of nonunion of the ulna. This conclusion is based on the fact that, first, union frequently fails to take place in the ulna and, further, that union following osteotomy is more likely if a bone graft, rather than a plate, is applied. In some cases, however, the fragments have united following osteotomy and the application of a vitallium plate. Fixation by either a bone graft or a plate is necessary in every case, since only by this means can recurrence of the deformity be prevented. If the deformity is of long standing or if the ulna is shortened, the head of the radius should be removed.

Permanent Dislocation of the Head of the Radius Without Deformity of the Ulna.—All our patients with this condition had sustained the injury during childhood and the head of the radius had not been reduced, or

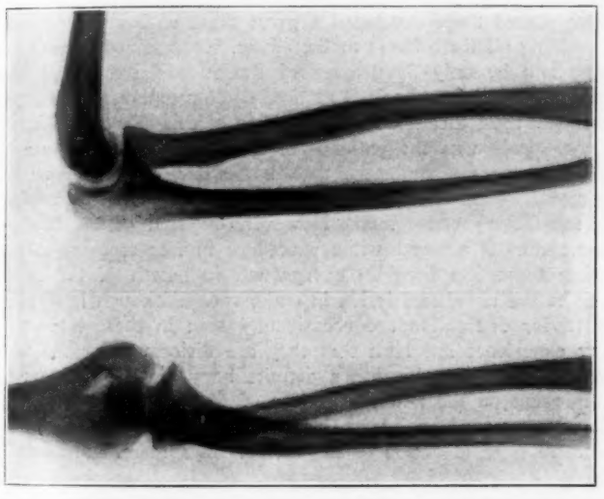

Fig. 11.—Lateral and anteroposterior views of same arm shown in figures 9 and 10 twelve years after reduction. The contour and function of the elbow are normal; the ulnar angulation has been spontaneously corrected with growth.

the dislocation had recurred. Although these patients have been too well satisfied with the functional results to desire treatment, it is conceivable that arthritic changes about the head of the radius or calcium deposits in the soft tissue might necessitate excision of the radial head during adult life.

END RESULTS

In adults, treatment of fractures of the upper one half of the ulna, 2 inches or more below the tip of the olecranon, by either closed reduction or open reduction and wiring of the fragments has resulted in radial angulation of the proximal fragment. Subluxation of the radial head has followed moderate angulation of the ulna, and complete dislocation has recurred from severe angulation. Some of these patients have obtained almost perfect function in the elbow; the majority, however, have limited motion, particularly on supination and pronation of the forearm. The impairment of rotation has varied from a negligible degree to practically total restriction, depending on the severity of the deformity of the ulna. On the other hand, the operative management herein advocated has effectually prevented angulation of the ulna and maintained reduction of the radial head; as a consequence the results have been gratifying from both an anatomic and a functional point of view.

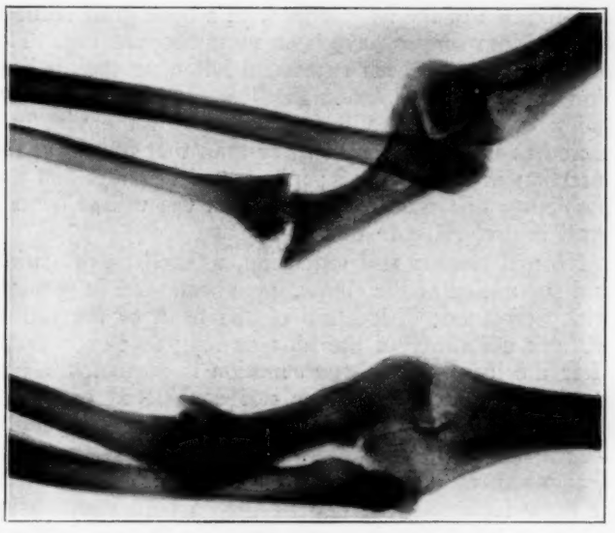

Fig. 12.—Lateral and anteroposterior views of ununited fracture of the ulna with dislocation of the head of the radius of twelve years' duration. The patient had previously had an open reduction and a resection of the head of the radius elsewhere.

In children, closed reduction has been more successful; in some cases, normal function of the elbow without deformity has been restored. In others, however, the dislocation of the head of the radius has recurred. The end results have been excellent following operative procedures similar to those advocated for adults. These procedures are recommended if there is any question as to accurate reduction of the head of

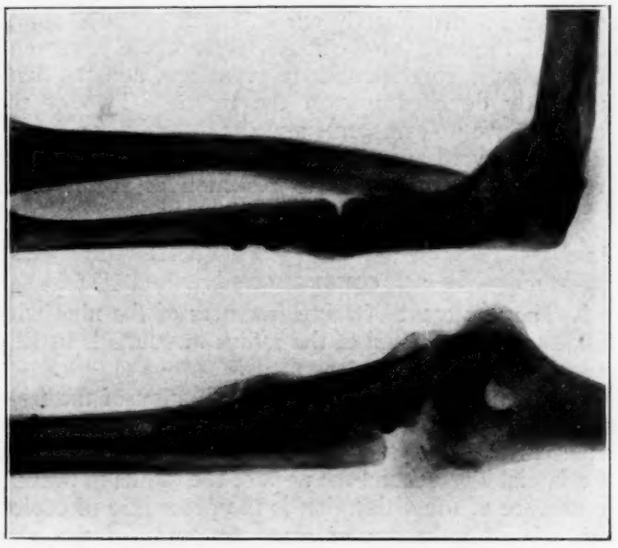

Fig. 13.—Lateral and anteroposterior views of same arm shown in figure 12 six months after the application of an onlay bone graft secured with vitallium screws, and further resection of the upper end of the radius. The ulna is clinically united and the patient is performing manual labor. Flexion of the elbow ranges from 45 to 170 degrees; motion is gradually increasing.

the radius or any tendency toward recurrence of the dislocation.

In the presence of nonunion of the ulna with dislocation of the radial head, good results have been obtained by the use of the onlay bone graft and fixation

by vitallium screws combined with resection of the head of the radius.

Treatment of malunited fractures of the ulna with dislocation of the head of the radius by osteotomy of the ulna and fixation with wire loops or other sutures has been disappointing, whereas the results of osteotomy and fixation of the fragments with a bone graft secured by vitallium screws have been most encouraging. The outcome has also been successful following the application of a vitallium plate. In these old cases, however, union of the ulna following the use of the vitallium plate has been distinctly slower than that following the insertion of a bone graft; in view of this fact and the possibility of nonunion in the ulna, the use of a bone graft is preferable to that of a plate.

Normal flexion and extension, as well as pronation and supination of the elbow, have been seen in patients with permanent dislocation of the head of the radius without deformity of the ulna as long as eleven years after the injury. Although motion is normal, the end results in these elbows are not considered excellent, since there is usually an increase in the carrying angle, muscle atrophy, weakness of the extremity, arthritic changes about the head of the radius, or a combination of these factors. One patient with a permanent dislocation of the radius had mild arthritic changes between the head of the radius and the humerus. On flexion of the elbow the radial head could be felt slipping over the lateral condylar ridge of the humerus, and crepitus was elicited when the forearm was pronated or supinated with the elbow flexed to 90 degrees. As this patient grows older the arthritic changes and symptoms may become sufficiently severe to justify removal of the head of the radius.

We have resected the head of the radius of fifteen patients. These patients have been followed over a period of years. Only three have developed any abnormality in the lower radio-ulnar articulation. These three have approximately one-half inch of radial shortening at the level of the radial styloid, the lower end of the ulna is correspondingly prominent and the hand is slightly deviated toward the radius. Two of the patients have no symptoms and the other has only mild pain in the wrist after unusual activity. Although there are theoretical objections to resection of the head of the radius, practically the procedure has not been of any material consequence.

CONCLUSIONS

1. The treatment of acute fractures of the ulna with dislocation of the head of the radius in adults is usually unsatisfactory by closed methods.

2. Open reduction with internal fixation of the fragments of the ulna by means of a vitallium plate and replacement of the radial head followed by the passage of a fascial loop around the neck of the radius to prevent recurrence of the dislocation is the procedure of choice.

3. Closed reduction of acute fractures of the ulna with dislocation of the head of the radius may often be employed in children; this, however, is frequently a hazardous procedure, as accurate replacement of the radial head is difficult and the dislocation is prone to recur.

4. For nonunion or malunion of the ulna with dislocation of the head of the radius, resection of the head and neck of the radius combined with an onlay bone graft of the ulna secured by vitallium screws is recommended.

5. Disturbances of the lower radio-ulnar articulation have not been of sufficient practical importance to contraindicate resection of the head and neck of the radius.

6. The operative technic in these fractures is simplified and danger of injury to the radial nerve is eliminated by use of the anatomic approach described.

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ABSTRACT OF DISCUSSION

DR. B. FRANKLIN BUZBY, Camden, N. J.: The authors' experience differs from mine in their frequency of open reductions of the dislocated radial head. I have always felt that in the care of this lesion, if the radial dislocation can be reduced, the ulna must of necessity be reduced, and the only disturbing element to be guarded against is the radial angulation of the ulnar fracture, which if present must be openly reduced and fixed. The ingenious incision advocated by the authors is well worth consideration since it does expose both lesions without endangering the radial nerve, and the use of forearm fascia is definitely an improvement ver the second incision in the thigh for fascia lata. A case illustrating some of the late bad results of this lesion, treated conservatively in the beginning, shows a divergent dislocation of both bones at the elbow, with a fracture of the ulna at its middle. The dislocation was easily reduced and held in flexion. Nonunion of the fracture ensued, which required late plating with vitallium screws and plate. As time passed there developed a great mass of bony overgrowth involving all three bones at the elbow, accompanied by no rotation and by only 30 degrees of flexion and extension, from 90 to 120 degrees. Lack of motion, while complained of bitterly, was not the indication for an excision of this elbow, which was done. Pain was the patient's chief complaint, and as can be seen by x-ray examination before the excision was done the removal of the head of the radius alone would not have given a satisfactory end result. However, excision of the head of the radius is a worth-while procedure in late cases especially, when there has been an overgrowth in length of the radius due to the unreduced lesion in childhood. Although lateral and posterior dislocation are occasionally seen in these late cases, my experience has been that they are dislocated for the most part anteriorly and cause a definite bony block to flexion by impinging on the anterior surface of the humerus. When the head is removed, the radius seems to fall into its proper position and, in spite of action of the biceps, stays there. In adults I have never found any disturbance of the lower radio-ulnar articulation after the excision of the head of the radius. Excision of the head of the radius in childhood, however, is a totally different matter and if at all possible should never be done before growth has ceased. When it is done, it is commonly followed in adult life by cubitus valgus, with secondary ulnar neuritis from stretching of the nerve in its normal groove. This of course can be relieved only by the transplantation of the nerve anterior to the internal condyle.

DR. HERMAN F. JOHNSON, Omaha: I wish to emphasize several points. It is frequently desirable to have x-ray examination of both elbows for comparative study, as a slight anterior position of the head of the radius is not easily recognized when examining the single joint. The necessity of open reduction and very rigid fixation of the shaft of the ulna to prevent the tendency to radial deviation is of prime importance. The approach as described by the authors has been worked out along anatomic lines and simplifies the operative procedure. This again brings out the desirability of the posterior approach to the elbow when extensive reconstructive procedures are necessary. Restoration of the annular ligament by the attached strip of deep fascia obtained from the upper forearm apparently is of adequate strength to maintain reduction of the head of the radius. I am rather reluctant to use Lane plates with multiple penetration of the cortex by screws, especially in compound fractures. However, with the use of the electrically neutral alloy vitallium the method is apparently a safe one. In my hands the use of long, slender steel pins, somewhat smaller than the Steinman pin,

passed down from the point of the olecranon process through the shaft of the ulna has proved to be the most satisfactory type of fixation. This pin is inserted through a short independent incision and is particularly desirable if the shaft of the ulna is comminuted. The base of the pin protrudes just enough so it can be grasped and thus easily removed when it has served its purpose. Drs. Speed and Boyd are to be commended on the excellent work they have done in this series of cases and the completed movie shown at their exhibit illustrates the approach very nicely and also shows some beautiful end results.

DR. G. MOSSER TAYLOR, Los Angeles: In reviewing the literature on this fracture, as the authors have said, the bulk of it is in foreign languages. The argument seems to be between two groups, one that emphasizes the necessity for securing the full length of the ulna, and the other group, which says that, if the radius is reduced, the ulna will fall into the proper alinement. The American writers seem to emphasize more the importance of open reduction for those cases in which the head of the radius is difficult to maintain in position. I want to present what I think will help us in relation to the paper that Drs. Speed and Boyd have given. In a closed reduction with the head of the radius not reduced as yet I used a beaded wire which held the head of the radius in place and lost a little position in the ulna, owing probably to a failure in technic. I don't think that I needed to have lost that. In the end result there is still angulation toward the radial side of the ulna. Two years later the doctor, now 45 years of age, has practically normal flexion and extension. He lacks a little in full extension, but rotation is lost about 50 per cent. The way in which I had to get most of this rotation was by manipulation. It took about five anesthetics and manipulation to force that rotation. Whether that is due to the adhesions about the head of the radius owing to the fact that the invaginated orbicular ligament was still held in its displaced position and the lesions following it had to be broken up, or whether that loss of rotation was due to adaptive shortening of musculature is a question. I believe that if this procedure of Dr. Speed and Dr. Boyd had been emphasized at the time this patient came in the man would now have a better range of motion.

DR. J. ALBERT KEY, St. Louis: This is a beautiful incision which makes the operation look a good deal easier than it is—at least than it has been in my hands in the few cases that I have done. Consequently I think it should be avoided when one is not competent to do this type of surgery. I believe that many of these cases can be reduced by the closed method and that the radial deviation of the ulna can be prevented by two wood splints incorporated in the plaster cast. These splints are padded. A long posterior and a short anterior splint are used. These are slightly wider than the forearm. They are put on the forearm and are pressed together so that they press the muscles between the bones and are strapped on with adhesive tape but not tightly enough to interfere with the circulation. The extremity is then encased in a plaster cast. Where competent surgery is available I think that a thorough débridement followed by implantation of sulfanilamide in the wound will permit the use of internal fixation with stainless steel or vitallium. The argument is still open as to whether or not vitallium has any advantage over stainless steel. I believe that the extra fixation given by the internal fixation plus the sulfanilamide is more of a preventive against infection than is the foreign body a detrimental factor.

DR. HAROLD B. BOYD, Memphis, Tenn.: We have successfully used a pin, as described by Dr. Johnson, in two cases of compound fracture of the ulna; however, we do not consider this to be ideal treatment for Monteggia fractures. I should like to emphasize further the angulation of the ulna toward the radius in these fractures. If the fracture is at its usual location (junction of the upper and middle thirds of the ulna), the pull of the supinator is enormous and the angulation, in our experience, cannot be prevented by external fixation. As much as I admire Dr. Key, I must say that I do not believe board splints are adequate to prevent the angulation which occurs in these fractures.

CANCER OF THE SKIN IN RELATION TO MULTIPLE MALIGNANT GROWTHS

SHIELDS WARREN, M.D.
AND
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Since the proposal has been made that the production of curable cancer of the skin by irritations which are not carcinogenic to internal organs may save some lives by preventing cancer of the internal organs, it becomes important to examine the data on which this proposal rests. On the basis of their figures, Peller and Stephenson [1] assume that cancer of the skin and lip exerts a beneficial effect in preventing the development of cancer elsewhere. So startling a suggestion as this deserves careful analysis. They show that in a total of 875,000 person-years of observation of United States Navy personnel there were ninety-one cases of cancer of the skin and lip with eleven deaths in a total of 113 cancer deaths.[2] Of these person-years of observation, over half (496,830) represented subjects below 30 years of age and hence not particularly useful for a study of cancer incidence. They contrast these figures with the cancer mortality calculated for similar age groups in the United States registration area for the period from 1928 to 1932 and for New York City for 1920. As is shown by their table, the figures are essentially the same for the navy personnel and for the United States registration area.[2] This fact they discard and contrast navy figures with those of New York City for 1920. They find 44 per cent fewer deaths among the navy personnel than would have been expected on the basis of New York City statistics for white men of corresponding age groups.

Previously Peller [3] had claimed, from calculations based on the occupational mortality statistics of England and Wales for the years 1921-1923, that an increase of carcinogenic irritation leads to an increase of cancer at the irritated spot but that this increase does not lead to an increase of total cancer mortality, causing rather a decrease because of a lessened incidence of cancer in more inaccessible organs.

In a careful survey Conrad and Hill,[4] using the basic data, showed that there was no inverse association between mortality from cancer of the skin and lip and mortality from cancer in other sites but that those occupations having a higher rate of skin and lip cancer have also an excess of cancer at other sites. This would fit in with our observation [5] that the person who has one cancer is definitely more likely to have a second cancer than would be expected on the basis of chance alone. In a recent study of multiple malignant tumors by Stalker and others [6] 167 cases were encountered in

Dr. S. O. Hoerr and S. L. Rea assisted in this study.
Read in part before the American Association for Cancer Research, March 19, 1940.
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1. Peller, Sigismund, and Stephenson, C. S.: Am. J. Hyg. (section A) **29:** 34 (Jan.) 1939; Am. J. M. Sc. (footnote 2).
2. Peller, Sigismund, and Stephenson, C. S.: Am. J. M. Sc. **194:** 326 (Sept.) 1937.
3. Peller, Sigismund: Lancet **2:** 552 (Sept. 5) 1936.
4. Conrad, K. K., and Hill, A. B.: Am. J. Cancer **36:** 83 (May) 1939.
5. Warren, Shields, and Gates, Olive: Am. J. Cancer **16:** 1358 (Nov.) 1932.
6. Stalker, L. K.; Phillips, R. B., and Pemberton, J. de J.: Surg., Gynec. & Obst. **68:** 595 (March) 1939.

which there was involvement of the skin or lip, and among these fourteen cases, or 8.4 per cent, showed malignant disease of other organs.

In order to present further data on this point we have surveyed the incidence of multiple malignant tumors in cases of cancer of the skin studied at the Huntington Hospital, Massachusetts State Tumor Diagnosis Service and the Pondville State Hospital for Cancer.[7] These cover 1,149 persons with basal cell or epidermoid carcinoma of the skin, followed for an

7. We have not included cases of cancer of the lip in our cutaneous cancer population because the difficulty of treatment of this lesion and its mortality are so much greater than those of cancer of the skin as to make any suggestion of induction of cancer of the lip for prophylactic purposes completely ridiculous.

TABLE 1.—*Age and Sex Distribution of Cutaneous Cancer Population According to Type of Cancer*

Classification of Data and Age Group	Men			Women			Total		
	Basal	Epidermoid	Total	Basal	Epidermoid	Total	Basal	Epidermoid	Total
20-29 years									
Number of cases	1	3	4	5	1	6	6	4	10
Average duration	8.5	0.5	2.5	3.7	8.5	4.5	4.5	2.5	3.7
Number of risk years	8.5	1.5	10.0	18.5	8.5	27.0	27.0	10.0	37.0
30-39 years									
Number of cases	17	8	25	16	2	18	33	10	43
Average duration	6.7	2.5	5.3	5.25	9.0	5.7	6.0	3.6	5.4
Number of risk years	113.5	18.0	131.5	84.0	18.0	102.0	197.5	36.0	233.5
40-49 years									
Number of cases	43	23	66	30	7	37	73	30	103
Average duration	7.7	6.5	7.3	7.5	8.6	7.7	7.6	7.0	7.4
Number of risk years	329.5	149.5	479.0	225.0	60.5	285.5	554.5	210.0	764.5
50-59 years									
Number of cases	100	44	144	59	26	85	159	70	229
Average duration	6.1	5.4	5.9	6.7	5.5	6.4	6.3	5.4	6.1
Number of risk years	610.0	236.0	846.0	398.5	142.0	540.5	1,008.5	378.0	1,386.5
60-69 years									
Number of cases	141	107	248	70	34	104	211	141	352
Average duration	5.8	4.7	5.3	5.7	4.9	5.5	5.8	4.8	5.4
Number of risk years	817.0	504.5	1,321.5	401.0	167.0	568.0	1,218.0	671.5	1,889.5
70-79 years									
Number of cases	94	117	211	56	42	98	150	159	309
Average duration	4.5	3.9	4.2	5.2	3.4	4.4	4.8	3.8	4.3
Number of risk years	426.0	453.5	879.5	292.0	143.0	435.0	718.0	596.5	1,314.5
80+ years									
Number of cases	32	37	69	11	20	31	43	57	100
Average duration	3.2	3.2	3.2	2.4	3.1	2.9	3.0	3.2	3.1
Number of risk years	103.0	118.5	221.5	26.5	62.0	88.5	129.5	180.5	310.0
Total									
Number of cases	429	339	768	248	133	381	677	472	1,149
Average duration	5.7	4.4	5.1	5.8	4.5	5.4	5.7	4.4	5.2
Number of risk years	2,424.0	1,481.5	3,905.5	1,449.0	601.5	2,050.5	3,873.0	2,083.0	5,956.0

TABLE 2.—*Occurrence of Multiple Cancers (Cutaneous and Internal) in Cutaneous Cancer Population*

Classification of Data and Age Group	Men			Women			Total		
	Basal	Epidermoid	Total	Basal	Epidermoid	Total	Basal	Epidermoid	Total
20-29 years									
Number of cases	0	0	0	0	0	0	0	0	0
Average duration	0	0	0	0	0	0	0	0	0
Number of risk years	0	0	0	0	0	0	0	0	0
30-39 years									
Number of cases	2	0	2	2	0	2	4	0	4
Average duration	1.5	0	1.5	2.5	0	2.5	2.0	0	2.0
Number of risk years	3.0	0	3.0	5.0	0	5.0	8.0	0	8.0
40-49 years									
Number of cases	2	5	7	4	0	4	6	5	11
Average duration	5.5	3.9	4.4	12.0	0	12.0	9.8	3.9	7.1
Number of risk years	11.0	19.5	30.5	48.0	0	48.0	59.0	19.5	78.5
50-59 years									
Number of cases	16	12	28	10	8	18	26	20	46
Average duration	5.8	4.6	5.3	7.8	7.0	7.4	6.6	5.5	6.1
Number of risk years	93.0	55.0	148.0	78.0	56.0	134.0	171.0	111.0	282.0
60-69 years									
Number of cases	32	33	65	13	5	18	45	38	83
Average duration	5.3	5.5	5.4	4.6	9.7	6.0	5.1	6.1	5.9
Number of risk years	169.0	181.5	350.5	59.5	48.5	108.0	228.5	230.0	458.5
70-79 years									
Number of cases	17	28	45	14	14	28	31	42	73
Average duration	2.6	3.8	3.3	4.6	2.4	3.5	3.5	3.3	5.8
Number of risk years	43.5	106.0	149.5	65.0	33.0	98.0	108.5	139.0	247.5
80+ years									
Number of cases	7	10	17	2	1	3	9	11	20
Average duration	2.5	3.1	2.9	0.5	0	2.2	2.1	3.3	2.8
Number of risk years	17.5	31.0	48.5	1.0	5.5	6.5	18.5	36.5	55.0
Total									
Number of cases	76	88	164	45	28	73	121	116	237
Average duration	4.4	4.5	4.5	5.7	5.1	5.5	4.9	4.6	4.8
Number of risk years	337.0	393.0	730.0	256.5	143.0	399.5	593.5	536.0	1,129.5

average period of 5.2 years or a total of 5,956 risk years, during which other cancers might have developed. By utilizing a cutaneous cancer population of this size we feel that errors due to chance have been more nearly eliminated than in the population utilized by Peller and Stephenson. We have not concerned ourselves with the incidence of cutaneous cancer in the total population, as our interest has been in weighing the validity of the suggestion made by them in 1939 that "the very low frequency of cancer of inner organs for the Massachusetts 1930 population as against an attack or morbidity rate of 1.29 ± 0.146 for our group of cases of cutaneous cancer. If we accept for the Massachusetts population a cancer attack rate of twice the cancer mortality rate, as has been suggested by Warren,[9] that gives a rate of 0.28 per hundred risk years (table 4) as contrasted with one of 1.29.

In every age group the observed number of cases exceeded the calculated number (table 3). In other words, the cancer attack rate for organs other than the skin in a population made up of patients with cancer of the skin is over twice as great as would be expected instead of showing, as suggested by Peller and Stephenson, a definitely lowered rate for cancer of the other organs.

TABLE 3.—*Occurrence of Cancer Other than Cutaneous Observed and Expected in Cutaneous Cancer Population*

Classification of Data and Age Group	Men			Women			Total		
	Basal	Epidermoid	Total	Basal	Epidermoid	Total	Basal	Epidermoid	Total
20-29 years									
Observed	0	0	0	0	0	0	0	0	0
Expected	0	0	0	0	0	0	0	0	0
30-39 years									
Observed	0	0	0	2	0	2	2	0	2
Expected	0.02	0	0.02	0.04	0.01	0.05	0.06	0.01	0.07
40-49 years									
Observed	1	1	2	1	0	1	2	1	3
Expected	0.25	0.11	0.36	0.35	0.095	0.445	0.60	0.21	0.81
50-59 years									
Observed	4	6	10	5	3	8	9	9	18
Expected	1.53	0.59	2.12	1.47	0.5	1.97	3.0	1.09	4.09
60-69 years									
Observed	8	11	19	6	2	8	14	13	27
Expected	5.08	3.11	8.19	2.50	1.06	3.56	7.58	4.17	11.75
70-79 years									
Observed	5	5	10	6	6	12	11	11	22
Expected	4.73	5.04	9.77	3.08	1.51	4.59	7.81	6.55	14.36
80+ years									
Observed	0	4	4	0	1	1	0	5	5
Expected	1.54	1.77	3.31	0.36	0.85	1.21	1.90	2.62	4.52
Total									
Observed	18	27	45	20	12	32	38	39	77
Expected	13.15	10.62	23.77	7.81	4.03	11.84	20.96	14.65	35.61

is explained as the result of the above mentioned increased skin irritation and Peller's concept of its role." [1]

The age distribution of the 1,149 cases by sex and type of cancer is shown in table 1. Of this group of patients with cancer of the skin, 237 developed additional cancers (of skin and internal organs) during the period that they were followed. One hundred and sixty-four subjects were men and seventy-three women (table 2). This incidence of multiple cancer represents 20.6 per cent of the cases. However, the tendency of cancer of the skin to be multiple has long been known and should excite no surprise.[8] When, however, we examine the figures for cancer of other organs than the skin associated with these cutaneous cancers, we find a total of seventy-seven, or 6.7 per cent (table 3). In every age group the cancer attack rate for organs other than skin exceeded materially that of the Massachusetts 1930 cancer death rate, being 0.14 per hundred risk years

TABLE 4.—*Cancer Attack Rate for Cutaneous Cancer Population per Hundred Risk Years*

Age Group	Massachusetts 1930 Cancer Death Rate per 100			Estimated Cancer Attack Rate per 100 Risk Years		
	Men	Women	Total	Men	Women	Total
20-29 years	0.01	0.01	0.02	0.02	0.02	0.04
30-39 years	0.02	0.05	0.03	0.04	0.10	0.06
40-49 years	0.08	0.16	0.12	0.16	0.32	0.24
50-59 years	0.25	0.35	0.30	0.50	0.70	0.60
60-69 years	0.62	0.64	0.63	1.24	1.28	1.26
70-79 years	1.11	1.06	1.08	2.22	2.12	2.16
80+ years	1.49	1.37	1.42	2.98	2.74	2.84
Total	0.12	0.16	0.14	0.24	0.32	0.28

SUMMARY AND CONCLUSIONS

On the basis of 1,149 carefully studied cases of cancer of the skin, we conclude that there is no justification for recommending the induction of cancer of the skin to protect against development of cancer elsewhere. In this population with cutaneous cancer there is definitely more cancer of organs exclusive of the skin than would be encountered in a similar population drawn from Massachusetts at large. Cancer of the skin does not protect against the development of cancer elsewhere. With this type of cancer as with cancer of the other organs, there is a greater number of multiple cancers than would be expected on the basis of chance.

TABLE 5.—*Cancer Attack Rate for Cutaneous Cancer Population per Hundred Risk Years*

Subjects	30-39 Years	40-49 Years	50-59 Years	60-69 Years	70-79 Years	80+ Years	Total
			All Cancers				
Men	1.52	1.46	3.31	4.92	5.12	7.68	4.20
Women	1.96	1.39	3.33	3.17	6.45	3.39	3.56
Total	1.88	1.44	3.32	4.40	5.55	6.45	3.98
			Not Skin Cancers				
Men	0.00	0.42	1.18	1.44	1.14	1.80	1.15
Women	1.96	0.35	1.48	1.41	2.76	1.13	1.56
Total	0.94 ±0.646	0.39 ±0.285	1.30 ±0.304	1.43 ±0.275	1.78 ±0.365	1.61 ±0.715	1.29 ±0.146

195 Pilgrim Road.

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INTRAMEDULLARY (STERNAL) TRANSFUSION OF HUMAN BONE MARROW

PRELIMINARY REPORT

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The unequivocal progress in the treatment of the deficiency anemias within recent years has also extended to the blood dyscrasias. The concept and subsequent proof of the absence of the intrinsic factor and consequent disturbance of the erythrocytic maturing factor substance has led to the successful treatment of pernicious anemia and other hyperchromic anemias. Iron therapy has yielded similar results in the hypochromic anemias. Certain anemias secondary to vitamin and endocrine disturbances have also yielded to therapy on the basis of the deficiency element. Supplying a specific deficiency seems to be the order of the day in the treatment of certain of the aforementioned anemias. A similar rationale might underlie the treatment of other blood dyscrasias. Such disorders as aplastic anemia, granulocytopenia, leukemia and thrombocytopenic purpura might well come under this category. If we assume that it takes erythrocyte maturing factor substance to cause the orderly conversion of the megaloblast into a normal red cell and so successfully treat pernicious anemia, the question arises why it is not possible to treat aplastic anemia or myelogenous leukemia by providing the patient with a similar "deficiency substance." In aplastic anemia the hematocytoblast would then function normally and give rise to a normal quota of megaloblasts, myeloblasts and megakaryocytes associated with normal maturative and delivery processes. In leukemia this unknown deficiency substance could afford the normal conversion of the myeloblast into a polymorphonuclear leukocyte.[1] In granulocytopenia the myeloblasts may be stimulated to function properly.

The next question that arises is that, if indeed there is such a factor, where does it reside? And if we know its residence, where in the patient would it do the most good? It was assumed that these substances would probably be found in the bone marrow and that they would exert their most direct benefit if they were introduced into the marrow. Experience with other deficiency dyscrasias has shown that there could be deficient absorption or utilization of the principle if given either subcutaneously or intravenously.

A new approach via bone marrow transfusion may be utilized in many ways. Where formerly liver extract, pentnucleotides,[1a] nucleinic acid[2] and "leukogens"[3] were given subcutaneously or intravenously, these and other substances can now be injected into the sternal marrow.[4] Yellow bone marrow extract,[5] given by mouth, has not achieved uniformly good results. Such substances as vitamins or iron and other medicaments may now be given by the intramedullary route in cases refractory to the same substances given in the usual way. Thus it may be possible to hold the key to the solution of certain blood dyscrasias as well as the more refractory of the anemias[6] or leukopenias.

It is interesting to record the method of bone marrow transfusion and recovery in a case of idiopathic aplastic anemia:

REPORT OF CASE

B. B., a white man aged 42, Jewish, a salesman of knitted goods, was admitted to the private service of Dr. Governale at the Long Beach Hospital on April 22, 1940, complaining of bleeding from the gums for the past seven days, painful swelling of the right lower jaw of three days' duration, and a general feeling of weakness and malaise for the past two weeks.

Since bleeding from the gums became more severe and nosebleeds of unusual severity occurred, and since the swelling of the right side of the face spread to such an extent that the patient was unable to open his mouth to partake of food, it was decided to admit him to the hospital.

The past history was negative except for the usual childhood diseases. Careful inquiry into the past revealed no history of any complaints requiring the use of any drugs (especially acetylsalicylic acid, aminopyrine, arsenic, arsphenamine or sulfanilamide and its derivatives), no exposure to x-rays or radium or the use of radioactive elements.

On admission the patient was pale and obese, and his temperature was 102.4 F. There was marked swelling of the right cheek involving especially the right lower jaw. The nostrils contained dry clotted blood. The mouth showed fresh and coagulated blood on the gums of the right cheek, associated with some tenderness in the region of the last molar. At this point there was some bleeding and black discoloration with evidences of an ulcerative and necrotic lesion. The soft palate and right anterior pillar also were involved by this lesion and patches of white exudate were seen nearby. Also the uvula and the base of the tongue were involved in this necrotic

From the Division of Hematology of the Departments of Medicine and Laboratories of the Jewish Hospital of Brooklyn.

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Yudina, N. D.: Changes in Blood and Hematopoietic System Following Introduction of Coal Tar into Marrow, Med. zhur. **6:** 63-68, 1936.
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Dessy, G.: Hematologic Changes Following Intramedullary Injections of Tar and Cancerogenic Carbohydrates: Study of Experimental Leukemia: Preliminary Report, Minerva med. **2:** 359 (Oct. 7) 1937.
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Benda, R., and Urquia, D. A.: Action of Intramedullary Injections of Iron and Other Substances in Experimental Anemia of Guinea Pig: First Results, Sang **11:** 891, 1937.
Lanza, G.: Myelogenous Leukemia Produced by Injection of 1:2 Benzopyrene into Marrow of Rats, Pathologica **30:** 185 (May 15) 1938.
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Fabiani, G.: Production of Schwartzman Phenomenon by Injections into the Marrow, Compt. rend. Soc. de biol. **130:** 1311, 1939.
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5. Marberg, C. M., and Wiles, H. O.: Yellow Bone Marrow Extracts in Granulocytopenia: Preliminary Report, J. A. M. A. **109:** 1965 (Dec. 11) 1937. Marberg, C. M., and Wiles, H. O.: Granulocytopoietic Fraction of Yellow Bone Marrow, Arch. Int. Med. **61:** 408 (March) 1938. Council on Pharmacy and Chemistry: Yellow Bone Marrow Concentrate (Armour Laboratories): Preliminary Report, J. A. M. A. **112:** 1257 (April 1) 1939.

6. Minot, G. R., in discussion on Rhoads, C. P., and Barker, W. H.: Refractory Anemia: Analysis of One Hundred Cases, J. A. M. A. **110:** 794 (March 12) 1938.

lesion. The posterior wall of the pharynx was not visualized because of the marked limitation of the movements of the jaw. Cervical nodes were not palpable. The heart and lungs were negative to percussion and auscultation. The liver and spleen were not palpable; the reflexes were normal. A roentgenogram of the lower jaw failed to reveal any abnormality of the right mandible. Possibilities considered on admission were subperiosteal abscess with perimaxillary phlegmon, osteomyelitis and granulocytopenia. The laboratory examination on admission revealed a hemoglobin of 58 per cent, red blood cell count of 2.64 million, white blood count of 2,300, and a differential which revealed polymorphonuclears 15 per cent, staff forms 5 per cent, lymphocytes 78 per cent, eosinophils 1 per cent, metamyelocytes 1 per cent, platelets diminished to absent, bleeding time six and one-half minutes and a coagulation time of two minutes. Urinalysis was entirely negative.

A transfusion of 500 cc. of compatible group A whole blood was given by the direct (syringe valve) method with no immediate reaction. However, there was a delayed one with a chill and rise in temperature to 104 F. The next day (April 23) there were no significant changes in the peripheral blood, but the urinalysis showed a trace of albumin and the presence of many red and white blood cells. Physical examination a few days later revealed no evidence of adenopathy or splenomegaly. Petechiae and purpuric spots were seen on the skin of the chest and abdomen. There was marked pallor but no evidence of an icteric tint. There was no heaping up of the gums. The buccal mucosa of the right cheek was the seat of a black, necrotic lesion in a serosanguineous bed. There was bleeding from the gums, especially of the right side.

Cytologic studies of the bone marrow and peripheral blood were then made. In the peripheral stream there were but few cells. It was impossible to count more than 10 cells on a smear. These included a few mature lymphocytes and occasional toxic polymorphonuclear leukocytes, a metamyelocyte and a myelocyte. All the white cells of the myeloid series seen on the smear were definitely toxic. The red cells showed evidence of marked regenerative effort with normoblasts, polychromasia, macrocytosis, anisocytosis and poikilocytosis. The platelets were markedly diminished in number. The examination of the bone marrow aspiration[7] revealed a hypocytologic marrow with no evidence of the myeloblastic picture of leukemia. The white cells consisted mainly of degenerated cells and smudges. The only significant features were the occasional clusters of erythroblastic cells arranged in a syncytium surrounded by intercellular matrix (fig. 1). It was then decided to introduce into the patient's sternal marrow the marrow of a healthy donor. This was done on the theory that it might supply some inherent hematopoietic quality which is present in healthy marrow but is absent in the marrow of the patient; i.e., to overcome a deficiency. The purpose of this procedure was to stimulate erythropoiesis, leukopoiesis and thrombocytopoiesis and thus overcome the panmyelophthisis in this case.

Accordingly a bone marrow aspiration was performed on the patient, and the needle with the solid obturator in it was left in situ. Then bone marrow was aspirated from the patient's brother (group O) and 3 cc. of bone marrow was withdrawn and immediately injected into the patient's sternal marrow. The next day (April 26) another transfusion of 5 cc. of bone marrow from the same donor was done. Previous therapy, including pentnucleotide, and liver extract intramuscularly, was discontinued and only small transfusions of 150 cc. of compatible citrated blood by the gravity method were given for a few days. Sulfanilamide was given for twenty-four hours in an attempt to stem the infective agent which might be the cause. This was discontinued because of the fear of further granulocytopenia. On April 27 another bone marrow transfusion of 5 cc. from another donor was performed.

Subsequently definite hematologic improvement was noted in the following order: The platelets appeared first (from one to two days later) and were easily found on all smears (originally diminished or absent on all smears). At the same time the bleeding tendency became less apparent. Finally when the platelets were normal in number (as shown in the table) all evidences of bleeding disappeared. Then the increase in the red blood cell count was noted. However, this increase was not significant. The rate of granulocytic increase was slower; this was manifested in that the white blood cell count rose from a low of 750 to 4,300 (fifteen days later) and to 7,000 (forty days later). This was striking in view of the fact that the lymphocytes diminished from a high of 82 per cent to a low of 23 per cent. This could be ascribed only to the neogenesis in the bone marrow, with the appearance of polynucleosis and a slight shift to the left of the white blood cells in the peripheral blood. The hemoglobin seemed to rise slowly and lagged behind the red blood cells. The patient received iron to stimulate further hemoglobin regeneration.

On June 3, forty-two days after admission, examination of the patient revealed no evidence of petechiae or adenopathy. The oral mucous membranes were normal in color and there was no evidence of bleeding from or heaping up of the gums. No exudates were seen and all traces of necrosis had disappeared. The liver and spleen were not palpable. The laboratory examination showed a hemoglobin of 73 per cent, red blood cell count of 5.0 million, white blood cell count of 7,000 and a differential which revealed polymorphonuclears 60 per cent, staff forms 6 per cent, eosinophils 3 per cent, lymphocytes 31 per cent and platelets adequate in number. Bone marrow aspiration revealed an increase of immature myeloid cells (probably hematocytoblasts or premyeloblasts), an increase in both eosinophilic myelocytes and polymorphonuclears

Transfusions and Results of Blood Examinations

Date	Transfusions: Blood	Transfusions: Bone Marrow	Hemoglobin, per Cent	Red Blood Cells, Million	White Blood Cells	Myelocytes	Metamyelocytes	Staffs	Polymorphonuclears	Eosinophils	Basophils	Monocytes	Lymphocytes	Platelets
4/22	500 cc.	.....	58	2.64	2,300	..	1	5	15	1	..	..	78	Diminished
4/23	500 cc.	.....	50	2.55	2,800	1	..	10	32	..	2	..	55	None
4/24	.....	.....	39	2.25	750	..	..	2	16	..	..	..	82	None
4/25	150 cc.	3 cc.	44.9	2.39	1,050	1	..	..	21	..	3	..	75	None
4/26	150 cc.	5 cc.	46.8	2.7	950	1	(10 cells seen)	2	..	..	1	1	4	None
4/27	100 cc.	5 cc.	35	2.45	850	..	(10 cells seen)	1	4	1	..	..	4	None
2/28	100 cc.	.....	34.5	2.52	1,400	1	(50 cells seen)	8	24	2	2	..	13	None
4/29	100 cc.	.....	35	2.54	2,400	..	..	2	22	4	4	..	68	Few
4/30	100 cc.	.....	36	2.42	2,600	..	2	4	38	..	2	..	54	Diminished
5/ 1	.....	.....	40.3	2.82	2,350	..	..	4	40	..	..	..	56	Normal
5/ 2	.....	.....	45.3	2.6	2,550	..	1	3	34	2	..	2	58	Normal
5/ 3	.....	.....	43	2.85	2,150	1	..	..	32	2	2	..	63	Normal
5/ 4	.....	.....	41.9	2.62	3,100	..	..	10	18	..	..	4	48	Normal
5/ 5	.....	.....	45.1	3.27	2,550	1	..	4	44	1	..	..	50	Normal
5/ 6	.....	.....	49.5	3.17	3,250	..	2	2	74	..	..	..	22	Normal
5/ 7	.....	.....	44.2	2.87	3,000	..	..	7	70	..	..	..	23	Normal
5/ 8	.....	.....	41.6	3.12	4,300	..	..	..	75	1	..	..	24	Normal
5/ 9	.....	.....	43.4	3.29	4,100	..	..	2	72	..	1	1	24	Normal
6/ 3	.....	.....	73	5.0	7,000	..	..	6	60	3	..	..	31	Normal

7. Morrison, Maurice, and Samwick, A. A.: A Simple Method for the Aspiration of Bone Marrow, J. Lab. & Clin. Med. **24**: 858 (May) 1939.

(a sign that betokens regeneration [8]) and an increase in megakaryocytes, reticulum cells and plasma cells.

Study of the bone marrow at this time showed that it had a close resemblance to that seen in leukemic myelosis. The marked erythroblastosis, G : E 50 : 50,[9] militated against this diagnosis. The marked increase of the hematocytoblastic elements associated with the mature white blood cells gave one the impression that regenerative effort was more or less successful. However, it was not justifiable at this time to credit these results solely to the injection of bone marrow. Further studies are necessary before conclusions can be drawn.

COMMENT

The patient presented a marked deficiency of all the components of the marrow and the rest of the cytologic inhabitants of the bone marrow. He showed a marked anemia, granulocytopenia, thrombocytopenia and an absolute lymphocytopenia. Because of the marked evidence of toxic polymorphonuclear leukocytes, degenerated cells and smudges, it was believed that a true panmyelophthisis was present.

The question of etiology brought up its relationship to drugs (aminopyrine, barbiturates, arsphenamines, sulfanilamide and its derivatives), radiation therapy, vitamin deficiency, contact with the aniline dyes and benzene derivatives. All these factors were carefully investigated and bore no relationship here. Included in the possible causes of this hematologic disorder must be considered leukemia of both varieties, malignancy, infection or sepsis. Leukemia was excluded because of the

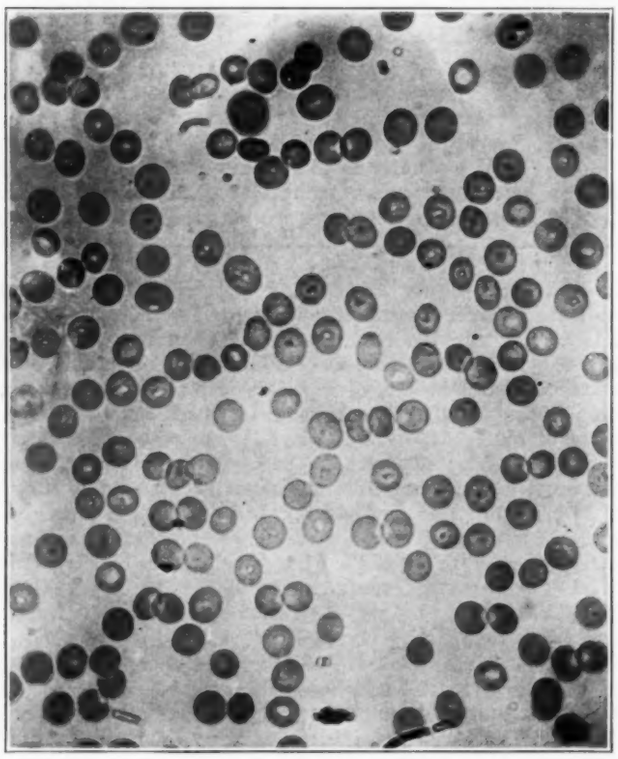

Fig. 1.—Peripheral blood, April 25.

lack of medullary myeloblastosis on three different occasions when bone marrow aspirations were done. Malignancy was ruled out by the clinical and hematologic course of the disease. Infection was the most likely because of the high temperatures and the inflammation present in the buccal cavity.

The cause and effect relationship is not clear because it is impossible to state whether the latter is secondary

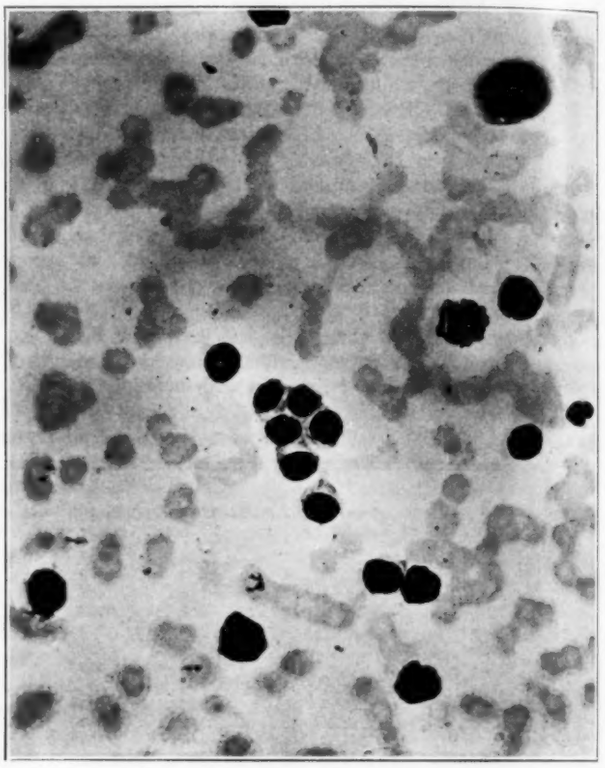

Fig. 2.—Bone marrow aspiration, April 25.

to the granulocytopenia or vice versa. One is therefore left with the conclusion that this case is one of idiopathic aplastic anemia.

Suffice it to say, at the present time, that human bone marrow may supply an important element inherent in healthy marrow but absent in aplastic anemia. One cannot attempt to guess what this factor is. It is tempting to suppose, from previous experiences in the development of hematologic disease entities such as pernicious anemia and hypochromic anemia, that there might also be a factor the lack of which is responsible for idiopathic aplastic anemia and bears the same relationship as the erythrocyte maturing factor in pernicious anemia and the iron (Fe) in idiopathic hypochromic anemia. The same concept may hold for granulocytopenia and purpura haemorrhagica.

The brilliant work of Osgood [10] on bone marrow culture led him to attempt the introduction of bone marrow culture intravenously on the supposition that the bone marrow would eventually take, but no opportunity was afforded for settling that assumption. An attempt was made by him to inject bone marrow intrasternally,[11] but, evidently for technical reasons, it was found impossible. It is interesting to note that it is a simple matter to introduce bone marrow intrasternally. One is surprised

8. Piney, Alfred: Recent Advances in Hematology, Philadelphia. P. Blakiston's Son & Co., 1927.

9. Morrison, Maurice, and Samwick, A. A.: Clinicohematologic Evaluation of Bone Marrow Biopsies, Am. J. M. Sc. **198**: 758 (Dec.) 1939.

10. Osgood, E. E.: The Histiogenesis, Classification and Identification of the Cells of the Blood and Marrow Based on Cultures and Hematological Studies of Human Marrow and Blood, Am. J. Clin. Path. **8**: 59 (Jan.) 1938.

11. Osgood, E. E.; Riddle, M. C., and Mathews, T. J.: Aplastic Anemia Treated with Daily Transfusions and Intravenous Marrow, Ann. Int. Med. **13**: 357 (Aug.) 1939.

with the ease with which the contents are expelled from the syringe with only the slightest amount of pressure. Two of our colleagues in other hospitals are following this method and have experienced no difficulty in several injections. At no time was there any evidence of superficial tumefaction, edema or ecchymosis which could belie the complete entry into the marrow cavity. An important point in the technic is the immediate introduction of the marrow to avoid coagulation. It has been noted that bone marrow coagulates rapidly when exposed to the air.

The question of injecting homologous or heterologous marrow is an interesting one. Following Osgood's hypothesis one would naturally favor the former. However, since one is introducing a "factor" for deficiency diseases, compatibility loses its significance but is nevertheless desirable. It has been found that there is no point in introducing excessive amounts of material obtained from the sternum because it has been noted that the first 1 or 2 cc. consists of true undiluted marrow while the taking of additional quantities is simply taking blood derived from the sinuses diluted with small quantities of bone marrow.

A method so simple affords the possibility of injecting elements or factors present in healthy bone marrow and may lend itself to the solution of many problems. According to Osgood, one of these is the problem of introducing growth centers or transplants. This hypothesis requires further confirmation. In view of our experiences in this and two other cases (chronic leukemic myelosis and anerythroplastic anemia) still being studied, it is felt that the more rational basis for success is a biochemical one supplying a factor analogous to the erythrocyte maturing factor or Fe. This brings up definite possibilities in combating various hematologic disorders. In leukemia, for instance, where the neoplastic and infective theories have been considered, one might envisage the deficiency of a certain substance which does not permit the myeloblast to mature and one might therefore be tempted in future cases to perform an "exsanguination" of the bone marrow in the sternum, immediately followed by the introduction of healthy bone marrow. This might cause the introduction of a factor that will initiate, either by a local or by a generalized effect, the metamorphosis of a resistant myeloblastosis to a polynucleosis.[1] The same type of procedure might be carried out for thrombocytopenic purpura and granulocytopenia. This method will therefore lend itself to diverse uses. This case is reported not so much for its particular use in aplastic anemia as for presenting a new modality in the treatment of blood dyscrasias secondary to a deficiency of certain hematopoietic factors or principles. The most aplastic bone marrow that can be visualized must always contain stem cells which should be susceptible of being stimulated provided the stimulus can reach the stem cell in the bone marrow. It is unnecessary to postulate that a take or growth of newly injected marrow alone is necessary to attain a successful result.

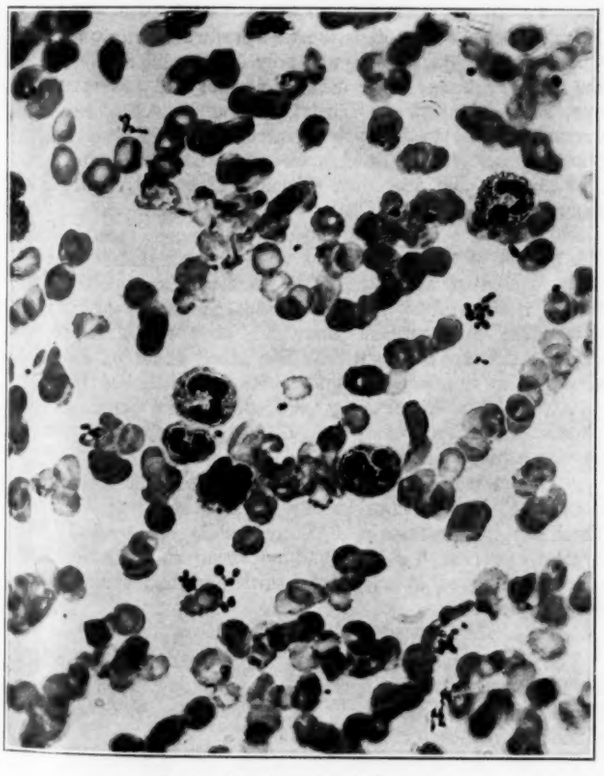

Fig. 3.—Peripheral blood, June 3.

Fig. 4.—Bone marrow aspiration, June 3.

CONCLUSIONS

1. In a case of idiopathic aplastic anemia, human bone marrow was introduced intrasternally.

2. An unknown factor present in normal marrow when thus injected may stimulate maturation of hematopoietic constituents already present in the diseased marrow, thus supplying a factor to overcome a deficiency disease.

3. Intramedullary (sternal) injections as a treatment is afforded by this method.

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Clinical Notes, Suggestions and New Instruments

CURE OF A CASE OF ACUTE ULCERATIVE ENDOCARDITIS

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Park Ridge, N. J.

Acute ulcerative endocarditis has been notoriously fatal. The prognosis was almost hopeless before the advent of chemotherapy. We report one case of acute ulcerative endocarditis which, because of the clinical cure, lends a more optimistic outlook in the treatment of this disease.

REPORT OF CASE

J. C., a white man aged 62, a sea captain in the merchant marine, complained chiefly of malaise and fever. The onset of the present illness occurred two weeks before he was seen, while he was returning from a port on the southern Atlantic seaboard, and was ushered in by an acute infection of the upper respiratory tract. It was accompanied by aches and pains throughout the body but particularly marked in the neck. There were general malaise and a dry, hacking, nonproductive cough. There were irregular episodes of chills and fever, and on several occasions the patient had recorded temperature rises to 104 F. He rapidly lost strength and was acutely ill when his boat came into port and he presented himself for medical attention. His past history was negative save for an episode of tobacco amblyopia more than twenty years before, from which he had recovered.

On physical examination, March 9, 1940, the patient was acutely ill; he was rather slender but well built. There was no cyanosis, dyspnea or edema, and no petechiae were noted. The temperature was 100 F., pulse rate 90 and blood pressure 130 systolic, 75 diastolic. Examination of the head was negative. The eyes, ears, nose and throat were normal. There was equal and adequate expansion of the thorax, and the lungs were clear to percussion and auscultation. The heart was moderately enlarged to the left on percussion and the apex beat was felt 2 cm. outside of the midclavicular line in the sixth interspace. There was regular sinus rhythm; there were no thrills or heaves. There was a soft, low pitched systolic murmur over the precordial region, with its maximum intensity at the apex. The murmur was not transmitted and it did not fill the entire systolic portion of the cycle. The liver was palpable at the costal margin but was not enlarged, tender or nodular. The spleen and kidneys were not palpable. There were no masses or tender areas in the abdomen or paravertebral regions. The genitalia and extremities were normal. The Wassermann and Kahn tests gave negative results. Urinalysis was negative. A blood count showed: hemoglobin 75 per cent, red blood cells 3,400,000, white blood cells 10,300; differential count: polymorphonuclear leukocytes 7.5 per cent, lymphocytes 13 per cent, monocytes 7 per cent, eosinophils 5 per cent.

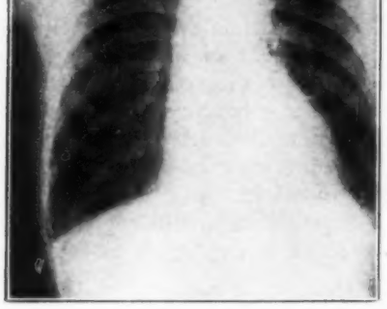

Fig. 1.—Appearance of the heart, March 9, 1940.

On x-ray examination both lung fields were completely clear. Oblique and lateral films were also taken to rule out any partially hidden pulmonary focus. The 6 foot heart plate showed moderate enlargement to the right and to the left. The aorta was tortuous and sclerotic, and several small calcific plaques were present in the knob. The transverse diameter of the chest was 29.5 cm. The transverse diameter of the heart was 16 cm., and the great vessels at the base measured 7.5 cm. The first and second oblique cardiac positions (R. A. O., L. A. O.) confirmed the tortuous, sclerotic nature of the aorta. On fluoroscopic examination there appeared to be normal cardiac pulsations.

On bed rest and antipyretics the patient continued to have a spiking temperature with fluctuations from 98 to 104 F. Agglutinations for Bacillus typhosus, Bacillus paratyphosus, Bacillus dysenteriae and Brucella melitensis were negative. Two blood cultures taken March 11 revealed pure cultures of beta hemolytic streptococci.

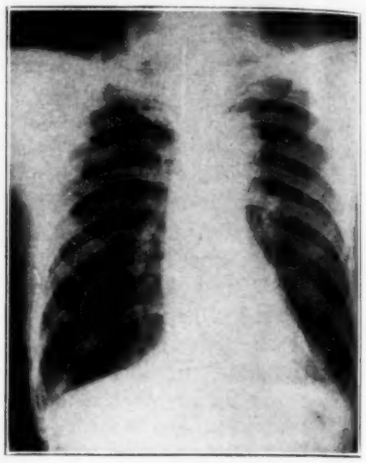

Fig. 2.—Appearance of the heart, July 7, 1940.

March 12 it was noted that the systolic murmur had become much harsher and now filled the entire systolic portion of the cycle. From this date the murmur was noted to change its character on several occasions. On March 16 the murmur was very harsh and loud. There was a distinct diastolic murmur extending from the apex up toward the aortic area.

Fifteen grains (1 Gm.) of sulfanilamide every four hours with an equal amount of sodium bicarbonate was started March 13. After ten days the temperature curve was decidedly smoother with daily swings to only 100 or 100.5 F. April 17 the patient's temperature became normal and has remained so ever since. During the second week of treatment the cardiac murmurs began to diminish in intensity and in their degree of transmission. In the third week only the systolic murmur was still audible, and this then vanished completely. At the last examination the heart sounds were excellent with no murmurs present. The patient became symptom free during the third week of treatment. The sulfanilamide dosage was gradually decreased after the fifth week and completely discontinued at the end of the sixth week. Six blood cultures taken subsequent to treatment have all been negative. Repeated urinalyses were all negative. The blood count by July 7 had returned to normal (hemoglobin 85 per cent, red blood cells 4,100,000, white blood cells 6,400; differential count: polymorphonuclear leukocytes 56 per cent, lymphocytes 33 per cent, monocytes 7 per cent, eosinophils 4 per cent).

A 6 foot heart plate on July 7 revealed that the heart had diminished in size considerably. The transverse diameter of the heart now measured 12.5 cm., while the shadows of the great vessels at the base had not been considerably altered. The patient was in excellent health and had returned to his ship command.

COMMENT

The diagnosis of acute ulcerative endocarditis is established by the changing heart murmurs, the positive blood cultures of hemolytic streptococci and the patient's septic course in the absence of other foci of infection.

The absence of embolic phenomena and splenomegaly is not unusual but rather the expected finding in acute endocarditis as contrasted with the manifestations in subacute bacterial endocarditis.

While it cannot be proved, it seems probable that the upper respiratory tract was the portal of entry for the infection. The success of therapy with sulfanilamide in this case may be related to two factors: Therapy was instituted very early in the disease, perhaps before deep bacterial invasion had occurred; secondly, the treatment was persisted in for a period of six weeks. There seems little doubt that moderate dosage,

long instituted, is more effective for deep seated infections than massive doses for a shorter period of time.

The striking improvement in the cardiac silhouette seems added evidence of cure in this case. The first cardiac measurements are those of a dilated heart secondary to acute valvular disease and septic systemic reaction. The second cardiac measurements represent a relatively normal heart, and the actual changes in cardiac volume are impressive. It is interesting to note the degree of dilatation that was present in this case with no signs or symptoms of failure of either the right or the left side of the heart.

SUMMARY

Cure in a case of acute ulcerative endocarditis due to Streptococcus haemolyticus followed moderate dosage of sulfanilamide given over a prolonged period of time.

12 South Main Street.

SUBACUTE BACTERIAL ENDOCARDITIS TREATED UNSUCCESSFULLY WITH SULFAPYRIDINE AND HEPARIN

ESTELLE E. KLEIBER, M.D., NEW BRUNSWICK, N. J.

This case is presented as one of subacute bacterial endocarditis in which treatment with sulfapyridine and heparin was unsuccessfully attempted. Unfortunately, this case had the fatal outcome usual in the vast majority of instances of this disease, but it is felt nevertheless that in the evaluation of a new form of therapy it is important to report the failures as well as the successes and to point out some of the practical difficulties in proceeding with a course of treatment theoretically desirable to stem the infectious process.

Efforts at treatment have been stimulated by the discovery of new chemical agents. Friedman, Hamburger and Katz [1] reported the use of heparin in the treatment of subacute bacterial endocarditis, this drug being chosen on account of its supposed capacity to prevent clot or thrombus formation. At the same time a report was published by Kelson and White [2] on the use of heparin in combination with sulfapyridine, and these authors were sufficiently impressed to urge further study of the combination of these drugs.

REPORT OF CASE

M. P., an Italian girl aged 19 years, was admitted to St. Peters Hospital Dec. 19, 1939, complaining of evening fever and painful joints. Her family history was essentially negative. Her past history revealed pleurisy and pneumonia at the age of 2 years with thoracotomy for empyema and frequent attacks of tonsillitis in childhood. Tonsillectomy was done at 16 years. Rheumatic fever with carditis occurred at 11 years and 16 years. She was admitted to the Cardiac Clinic at Middlesex Hospital, where she had been observed from March 1939, her chief complaint being cough, dyspnea, orthopnea and occasional precordial pain. She was found to have active rheumatic heart disease with mitral stenosis and insufficiency, cardiac enlargement and tachycardia. The PR interval was 0.22 second. The patient was advised to continue bed rest; she was put on a high caloric diet with additional vitamins, and digitalis intake was temporarily reduced. Salicylates were given for symptomatic relief. Her temperature was normal on every clinic visit. From March 1939 until her admission in December to the hospital the patient was not under medical supervision.

TABLE 1.—*Sulfapyridine Concentration*

Date	Mg. per 100 Cc.
1/11/40	2.5
1/13/40	0.5
1/16/40	6.7
1/17/40	3.1
1/18/40	2.5
1/19/40	2.5
1/21/40	6.5
1/22/40	8.2

On admission to the hospital the patient gave a history of having had a fever and cough for one month, diagnosed as grip by a physician called into the home. She had mild joint pains and occasional precordial pain radiating to the back. On physical examination her condition was similar to that just related with certain important additions: (1) fever of 100 F., (2) tender spleen palpable three finger breadths below the left costal margin, (3) tenderness of the finger tips, (4) more pronounced pallor and (5) petechial hemorrhages in the conjunctivas and on the extremities.

The blood count on admission showed hemoglobin 70 per cent, red cell count 3,950,000, white cell count 5,900, polymorphonuclear leukocytes 72 per cent, lymphocytes 23 per cent, monocytes 4 per cent and eosinophils 1 per cent. During the sulfapyridine therapy, blood counts were repeated daily and showed very little variation except for a transient rise in the white cell count to 16,800. The only significant change in the hemoglobin content occurred Jan. 23, 1940, when she had had a frank hemorrhage; then the hemoglobin content was reduced to 42 per cent with 2,740,000 red blood cells. Following transfusion, the values remained between 55 and 66 per cent, with a red blood count of 3,800,000. Sulfapyridine concentrations are shown in table 1 and the results of blood cultures in table 2. Blood chemistry January 23 showed uric acid 3.3 mg. per hundred cubic centimeters; February 1 urea nitrogen was 11.2 mg.; March 22 urea nitrogen was 10.5 mg. and sugar 81.5 mg. The Kline test gave negative results. The sedimentation rate Dec. 20, 1939, was 12 mm. in one hour and five minutes. The circulation time March 8, 1940, was eleven seconds.

On admission the urine showed an acid reaction, a specific gravity of 1.022, a trace of albumin and an occasional white blood cell. X-ray study of the heart confirmed a previous fluoroscopic examination suggestive of mitral stenosis. It also showed a slight accentuation of bronchial markings, especially on the right side. The temperature during the first few weeks fluctuated between 100 and 102 F.; thereafter it became more spiking in character and reached a much higher average elevation (104 F.), except for certain variations to be noted.

After the first positive blood cultures on Jan. 5 and 8, 1940, it was decided to start medication with sulfapyridine together with heparin in an effort to prevent further vegetations. The patient was given 30 grains (2 Gm.) with 20 grains (1.3 Gm.) of sodium bicarbonate repeated in four hours, with subsequent doses of half this size every four hours. Ascorbic acid (200 mg.) was administered daily for three days and 100 mg. daily thereafter. The patient tolerated the drug poorly, vomiting after each dose, but her temperature became normal for the first time since admission.

On January 11 sulfapyridine concentration of the blood was only 2.5 mg., and a sharp pain developed in the region of the bladder and dull pain with tenderness over the right kidney. The urine showed many red blood cells; this may have been due to sulfapyridine or more likely to embolic glomerulonephritis. Because of the gastrointestinal upset sulfapyridine medication was attempted by rectal suppositories, but the blood level could not be raised above 0.5 mg. in this manner and therefore on January 16 oral medication was resumed and was much better tolerated. The concentration in the blood now rose to 6.5 mg. and no further red blood cells were present in the urine. January 17 the patient passed two reddish brown calculi following an episode of renal colic. These stones consisted mostly of uric acid, though a few sulfapyridine cystals were found in them. On January 19 heparin was started by continuous intravenous drip, 10,000 units to every 500 cc. of physiologic solution of sodium chloride. Bleeding and clotting time were determined daily during this period but no significant changes were noted. The urine again became bloody and

1. Friedman, Meyer; Hamburger, W. W., and Katz, L. N.: Use of Heparin in Subacute Bacterial Endocarditis, J. A. M. A. **113**: 1702 (Nov. 4) 1939.
2. Kelson, S. R., and White, P. D.: A New Method of Treatment of Subacute Bacterial Endocarditis, J. A. M. A. **113**: 1700 (Nov. 4) 1939.

after a total of 90 cc. of heparin (900,000 units) had been given gross hematuria started and several uric acid stones were passed. Her pallor increased and the cough became severe. Heparin and sulfapyridine were both discontinued because of the progressive anemia, weakness and bleeding. Several small transfusions were given. On January 31 a cerebral embolus developed. Blood culture showed more than 200 colonies. Sulfathiazole then was given temporarily but failed to change the picture. Thereafter her course was steadily downhill till her death on June 5 with right hemiplegia.

At autopsy the diagnosis was confirmed and on microscopic section some healing was found in the vegetations on the mitral valve and auricle. Multiple infarcts were present in the spleen and kidneys.

COMMENT

This case presented a known continuous rheumatic heart infection for at least a year and a half previous to the onset of the present illness, as shown by continuous tachycardia, frequent precordial pain, accelerated sedimentation rate and a prolongation of the PR interval in the electrocardiogram. It presented, therefore, suitable soil for the implantation of a bacterial endocarditis. This condition was confirmed soon after the patient's admission and a plan of treatment was determined.

Certain practical difficulties presented themselves almost immediately after treatment with sulfapyridine was started. The drug proved to be toxic to the patient in that she was rendered continuously nauseated and vomited frequently during the early days of its administration. This made it difficult to maintain a suitable level of blood concentration, nor could the latter be maintained by a change to rectal administration. Even more troublesome than this problem was that of the increased tendency to bleeding, induced probably by the intravenous administration of heparin. Three days after the beginning of sulfapyridine therapy the patient first experienced renal pain and passed several calculi. To our surprise, the stones consisted chiefly of uric acid with a few crystals of sulfapyridine, which may have served as a nucleus in the further deposit of the other material.

TABLE 2.—*Blood Culture*

Date	Culture
1/ 5/40	30 colonies per cubic centimeter of Streptococcus viridans after 48 hours
1/ 8/40	30 colonies per cubic centimeter of Streptococcus viridans after 48 hours
12/20/39	30 colonies per cubic centimeter of Streptococcus viridans after 3 weeks
12/22/39	30 colonies per cubic centimeter of Streptococcus viridans after 3 weeks
1/23/40	Negative
1/26/40	Negative
2/ 4/40	Negative
2/19/40	Approximately 200 colonies of Streptococcus viridans per cubic centimeter

Gross hematuria became a major problem only after the administration of heparin. In spite of the fact that the coagulation time was not markedly changed, the patient's condition became so alarming that it was necessary to omit both sulfapyridine and heparin in order to combat the progressive anemia. As can be seen by the laboratory observations in table 2, three negative blood cultures were obtained after the administration of sulfapyridine had resulted in adequate blood concentration of the drug, and it was only following the cessation of this treatment that cultures again became positive. It is of interest that at autopsy some evidence of healing in the vegetative processes was found in the auricle and on the valves.

SUMMARY

1. In a proved case of subacute bacterial endocarditis sulfapyridine and heparin were given and the blood cultures were temporarily rendered sterile.

2. Severe hematuria necessitated the cessation of treatment.

3. Evidence of healing of some vegetations was found at autopsy.

131 Livingston Avenue.

NAIL LACQUER DERMATITIS

LESTER HOLLANDER, M.D., PITTSBURGH

Three cases of nail lacquer dermatitis came under my observation showing a similarity of clinical signs and therefore considered worthy of record:

CASE 1.—Mrs. E. E., aged 26, an American housewife, presented herself on account of an eruption of six months' duration, which affected the upper eyelids, the face about the chin, two sharply defined plaques on the left side of the neck under the chin and areas about the hairline and the nucha. Itching and burning were severe enough to awaken her during the night; although annoying during the day, they were most acute at night and on arising.

Cosmetics were suspected by the dermatologist who attended her, but since patch tests to powders and creams were found to be negative a diagnosis of neurodermatitis was made. She was treated chiefly with x-rays, which would ameliorate the symptoms, but then the condition would return with intense violence.

When she came under my care she was ordered to discontinue all of her cosmetics and within four days a marked improvement was noted, giving the first corroboratory evidence that I was dealing with a cosmetic dermatitis. After a period of two weeks she was allowed the use of a single cosmetic each week. The first week she was permitted the use of a lipstick. The second week face powder was added. The third week she was permitted the use of nail lacquer. Within forty-eight hours the two peculiar plaques on the left side of the neck reappeared and the upper eyelids became reddened. It was easily discerned then that the nail lacquer was the cause of the trouble, and the manner of its production was also apparent. The patient, by doubling her hands, kept her nails against her neck during her sleep. She slept on the left side and she used her hands as a support.

Discontinuation of the nail lacquer was followed by disappearance of all her symptoms.

CASE 2.—Miss A. M. P., aged 21, an American, presented herself on account of an eruption which affected her eyelids and chin. This eruption would come and go and was of one year's duration.

Patch tests were made on her with all her cosmetics and all the tests gave negative results. However, she was placed on the regular removal and replacement routine followed in these cases, and the irritant was thus found to be the nail lacquer. In this case also the discontinuation of the use of nail lacquer was followed by complete recovery.

CASE 3.—Mrs. H. McV., aged 32, a beauty shop operator, presented herself on account of a marked dermatitis of the upper eyelids and several areas about the chin. The condition was a recurring one and of about two years' duration. Itching and burning were annoying symptoms, chiefly at night. Without any apparent reason the eruption would fade and then, apparently without any reason, it would light up again, each time to be more pronounced. She definitely stated that her beauty shop had nothing to do with it as these areas of dermatitis were worse during her vacation periods, which, of course, were spent away from the beauty shop.

The elimination of cosmetics was a more serious problem with this patient. However, after several trials at guessing at the cause, each of which failed, the patient was persuaded to stop all her cosmetics. The permission to use nail lacquer was followed by the recurrence of the upper eyelid eruption and faint signs of dermatitis about the chin. Discontinuation of nail lacquer cleared this troublesome contact dermatitis.

COMMENT

The points of similarity in these three cases may be taken up under several headings:

1. There was a similarity of the involvement of the affected areas. In each case the eyelids and areas of the chin below the lower lip were involved. This must have occurred by contact, such as straightening the eyebrows with the lacquered nails and possibly by the habit of playing with the areas on the chin. In case 1 the peculiar distribution on the left side

From the Pittsburgh Skin and Cancer Foundation.

of the neck has been explained. This particular involvement handicapped one's suspicion of the real offending agent. One would imagine that nail lacquer dermatitis would affect the nail folds or perhaps the palmar surfaces, chiefly those of the thumbs. None of this involvement was present.

2. In each instance the patch testing gave negative results, even with the offending agent. This can be explained only on the presumption that sensitivity of the affected areas occurred after repeated contacts and that this sensitivity was cultivated.

3. Subjective symptoms were noted chiefly at night. This is explained by the hypothesis that it was during the sleeping period that the nail lacquer came in contact with the affected areas more continuously and more frequently.

4. A word of explanation is needed in describing the method which I prescribe when the use of cosmetics is interdicted. The patient is ordered to shampoo her hair or to have some one shampoo it at home in order to remove waveset, hair oil, hair tonic, hair dressing, scalp ointment or any of the various types of so-called rinses which are used to bring out sheen or color of the hair. After the shampoo the hair is rubbed dry with a turkish towel. The patient is instructed to cover her hair with a large kerchief before retiring so that if by some chance any of the aforenamed cosmetics should remain attached to the hair shaft they would not become operative again as irritants or sensitizers.

All creams—cleansing, foundation, tissue, cold and vanishing—are ordered discontinued. The patient is instructed to wash her face with a certain nonscented soap so that one can be sure of the absence of perfume. The nail lacquer is ordered removed. Sachet powders, perfumes, lipstick, eye shadow, eyebrow pencil, eyebrow and eyelash ointments, artificial lashes and artificial nails are interdicted each by name. It is only after the recital of each of these and the thorough understanding by the patient of the word "cosmetic" that one may be reasonably certain that the instructions will be followed.

5. In two of the three cases a dermatitis caused by cosmetics was suspected by the attending dermatologists. The peculiar distribution of the eruption and the misleading patch tests threw both of them off the proper diagnostic track.

6. I should like to mention the great dissimilarity of the cutaneous textures of these three patients. The first was a brunette; her skin was thickened and oily. The second patient was a very light blonde with markedly freckled and satin-like skin. The third patient was a brunette with normal cutaneous structure, showing that sensitivity, at least in this group, had nothing to do with skin structure.

CONCLUSION

The manifestations of three cases of nail lacquer dermatitis occurred on particular areas of the face, and there was a total absence of cutaneous symptoms about the nail folds or other parts of the hands. It is reasonably safe to assume that on account of this distribution some cases go unrecognized. Therefore emphasis must be made of the fact that in reality the symptoms of contact dermatitis should be logically looked for about the face, since nail lacquer, acting either as an irritant or as a sensitizer, will come in contact with the face more frequently than with any other of the exposed cutaneous surfaces.

Look for nail lacquer dermatitis on the face.

631 Jenkins Building.

Self Reliance.—A key to the mental hygiene of early childhood lies in building up adequate self dependence. Even in infancy this principle of self dependence must be respected. Not only from the breast must the child be weaned. By slow gradations he must develop fortitudes which lie at the basis of detachment. He cannot always play in his mother's lap; he must in time begin to play in his pen. He cannot always play in the same room with his mother; he must learn to play in an adjoining one. . . . These are elementary lessons in self reliance, but the detachment must not be hurried, and all along the path of preschool development our demands should be tempered to meet the child's immaturity.—The First Five Years of Life—A Guide to the Study of the Preschool Child, New York, Harper & Brothers Publishers, 1940.

Special Clinical Article

THE MANAGEMENT OF PATIENTS WITH HEART FAILURE

CLINICAL LECTURE AT NEW YORK SESSION

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Amid the national discussion concerning medical practice and the care of the sick it is well for members of the medical profession and the nonmedical public as well to realize that the treatment of disease has improved greatly during the past generation. It is obvious that new therapeutic discoveries such as insulin, liver extract, sulfanilamide and other specific remedies have saved many lives. But what is not so clearly understood is that old remedies are now more intelligently and more effectively applied, with the result that suffering is diminished and life is prolonged. This is particularly true of heart disease. Even in the short span of twenty years I have witnessed a striking improvement in the care of patients suffering from cardiac disorders in general practice. We may bewail the passing of the old family practitioner and the loss of his service in the civil affairs of his community and his wise counsel in many domestic problems that he helped solve but we need not lament about the character of the medical service that the average citizen is receiving at the hands of the present family physician.

From fifteen to twenty years ago a majority of the patients with congestive heart failure that I saw in consultation could be helped considerably and in many instances could be rehabilitated to a useful life. They had not received the benefit of the known therapeutic procedures then available. They had not been given adequate digitalis or diuretics, an extensive hydrothorax went undetected, an underlying thyrotoxicosis was overlooked or some other error in diagnosis or treatment accounted for the failure to recover compensation. Now, as a result of the superior training and the greater dissemination of present knowledge, it is the exceptional case in which a consultant is able to improve greatly the outline of treatment as prescribed by the practitioner and then it is more apt to be the result of a more accurate diagnosis. For this reason the first step in the proper care of a failing heart is a correct diagnosis.

This discussion will be confined to the treatment of heart failure, often called congestive heart failure, and is not meant to include the treatment of angina pectoris or acute coronary thrombosis. At the outset it must be clear that many conditions still confused with heart failure are, in fact, instances of peripheral circulatory failure or of simple tachycardia and that the treatment that is effective in the one will be entirely valueless and at times harmful in the other. The rapid pulse, cold extremities, asthenia and collapse frequently seen in infections like pneumonia or in postoperative shock are not due to heart failure. Similarly many patients with dyspnea are suffering from chronic bronchitis, emphysema, tumor of the lung, aneurysm of the aorta or other noncardiac disorders. In fact, there are numerous instances in which breathlessness is due merely to

From the Medical Clinic of the Peter Bent Brigham Hospital and the Department of Medicine, Harvard Medical School.

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obesity, general weakness, anemia, psychoneurosis or hysteria. Even swelling of the legs may be misinterpreted as an evidence of heart failure when the cause is varicose veins, phlebitis or hypoproteinemia. Furthermore, enlarged liver and ascites, so commonly the result of heart failure, may be confused with cirrhosis of the liver and abdominal neoplasms. It is obvious that we must start with an accurate diagnosis and know that the heart itself is at least in part responsible for the symptoms that we are treating.

Another difficulty that arises is the distinction between heart disease and heart failure. Although it may be generally assumed that the latter cannot be present without the former, patients often have heart disease for a great many years without evidence of failure. During these years they may be quite well or have functional symptoms or present themselves with symptoms of other noncardiac disease. It is obvious that treatment directed at heart failure when a structurally diseased heart is well compensated will not only be useless but may be harmful. Accurate diagnosis is therefore imperative not only in determining the presence or absence of organic heart disease but in estimating the degree of cardiac efficiency.

When there is doubt as to whether the symptoms or signs in a given case are due to heart failure or to some other condition, it may be necessary to investigate the problem by means of special laboratory procedures. It is not my purpose here to discuss the various examinations there may be or, in fact, need to be carried out to make this differentiation. Suffice it to say that occasionally one is compelled to measure the velocity of blood flow, the venous pressure, the vital capacity of the lungs, the blood volume or other aspects of the circulation in order to decide whether a given patient has or has not congestive heart failure.

Having concluded by one means or another that a given patient has congestive heart failure, let us trace the plan of treatment. The measures to be employed and the time spent in the entire program will naturally depend on the severity and the individual peculiarities of the case. Among these factors must be considered the economic and psychologic circumstances involved. It is obvious that matters such as special nurses, a prolonged vacation or a sea voyage may be intelligently employed in one case and be prohibitive in another. These are questions that should be carefully considered with due regard to the expense and the results to be expected.

Let us assume that we are confronted with a patient in average financial circumstances who has early congestive failure on the basis of hypertensive heart disease. The treatment to be discussed will not be materially different whether the underlying cause is valvular disease, coronary artery disease or syphilis. The prognosis or response to treatment may differ but, except for specific conditions to be discussed later, treatment will be the same.

REST

Of primary importance is rest. Except for the mildest cases, in which merely curtailment of some activities or the institution of rest periods in the afternoon or over week ends will suffice, it is advisable to have the patient rest at home or in a hospital. It often proves to be a waste of time to compromise with half measures or to send the patient away for a vacation which is apt to be both expensive and worthless.

We have been taught that the heart obtains its maximum rest with the patient in bed twenty-four hours a day. Although this is generally true, certain clinical experiences viewed in the light of recent experimental observations necessitate reconsideration of this very important question. All physicians are familiar with the story some patients tell us that they feel respiratory distress only at night. Nocturnal dyspnea and suffocation is common in conditions accompanied by primary left ventricular strain. Patients with hypertension, coronary artery disease, aortic valvular disease and less frequently those with mitral stenosis may have attacks of nocturnal breathlessness in bed and yet feel quite comfortable while up and about during the day. It is surprising that until the present, on instituting a course of treatment, we have insisted that such patients should be put to bed. I know that at times I have urged such a course when the patient himself would plead to be permitted to remain up and about or at least to sit in a chair. In most cases when such patients are put to bed and a suitable medical regimen for heart failure is carried out, clinical improvement takes place and compensation is restored. In some instances, however, patients who do not appear to be in a critical condition steadily go down hill and die. We can all recall experiences in which it seemed that from the time the cardiac patient was put to bed the condition grew worse. It is my purpose to throw some light on this apparent paradox and to try to define the limitation of strict bed rest treatment.

When patients are put to bed there result some simple changes in the dynamics of the circulation. It is obvious that, with the legs elevated or horizontal, return flow of blood or fluid from the periphery is facilitated. This tends to increase the filling of the right side of the heart and the lungs. Under normal circumstances the increased inflow into the right side of the heart is counterbalanced by an increase in the output of each ventricle and no troublesome stagnation develops. But if the left ventricle is already overburdened and cannot keep pace with the increase in blood to be expelled or if there is a sufficient obstruction in the mitral valve the added blood may be partially trapped in the lungs, resulting in congestion or pulmonary edema. It is known that normally [1] there is a decrease in the total lung volume of over 300 cc. and a decrease on the vital capacity of the lungs of about 200 cc. in the recumbent position. Very likely these figures are even greater for patients already suffering from pulmonary congestion. These changes must indicate an increased congestion of the pulmonary vessels. Not only is the limited breathing space further reduced but the congestion of the lungs acts as a trigger mechanism through reflexes in the production of paroxysm of dyspnea. Although there are other factors in the production of dyspnea, the aforementioned mechanism plays a significant role.

These single observations serve to explain some common clinical observations. It frequently happens, when an ambulatory patient who primarily complains of dyspnea is put to bed, that during the first few days pitting edema of the legs disappears. Simultaneously in some cases evidence of pulmonary congestion and dyspnea increases. Some of the peripheral fluid has shifted into the lungs. This may occur even when no pitting is detected, for cardiac patients can have several liters of tissue edema without obvious pitting. Now such a patient may be in a much worse condition, for fluid in the legs does but little harm whereas additional

1. McMichael, John, and McGibbon, J. P.: Postural Changes in the Lung Volume, Clin. Sc. 4: 175 (Nov. 2) 1939.

fluid in the lungs may be dangerous or even fatal. I have often detected hydrothorax develop in patients after being put to bed who previously had only a few moist basal rales. The foregoing is an indication of deleterious change in the dynamics of the circulation. I have recently observed a fall of the vital capacity of the lungs during the first day or two after instituting bed care in some of my cases.

The reason that harmful results from strict bed rest are not encountered more frequently is that in most cases the beneficial effects of rest, diet and medication outweigh the harmful effects of posture. The improvement in the efficiency of the heart is great enough to keep pace with the increase in return flow. However, in a small group of cases when such preliminary improvement does not or cannot take place the effect is harmful and may be disastrous. The danger is greater in older patients, in those with serious heart failure, and especially when there is paroxysmal nocturnal dyspnea.

The inference from the foregoing is that, although complete bed rest is often desirable in the treatment of heart failure, careful observation is necessary to see that the very symptom we are treating, i. e. breathlessness, is not made worse. There will be times when one should deliberately keep patients in a chair to avoid this danger or to undo the harm produced by the recumbent position. Patients with paroxysmal cardiac dyspnea have been treated successfully by medication even while being allowed to remain ambulatory.[2] I feel quite certain that I have occasionally saved a patient's life by taking him out of bed and allowing him to sit in a chair with his feet hanging down. The following experience is illustrative:

A shopkeeper aged 58 had a typical attack of acute coronary thrombosis. During the three weeks following the onset, severe increasing dyspnea developed. When I saw him he was somewhat irrational, having marked orthopnea and Cheyne-Stokes breathing. The lungs were full of rales but there was no peripheral pitting edema. He had been in bed under an oxygen tent most of the time. I advised taking him out of bed, to discontinue the various medication such as aminophylline and to place him in a chair with his feet hanging down. Within a few hours his condition began to improve. Two days later he showed edema of the lower legs but the orthopnea was practically gone. He then received digitalis and mercupurin, had a satisfactory diuresis and gradually recovered compensation. He has been ambulatory and in fairly good health the past three years. The turning point in this patient's condition occurred directly after getting him out of bed into a chair.

Now we may return to the hypothetical patient who is being put to bed. It generally requires a period of three or more weeks, depending on individual factors. The bed should be made comfortable with proper pillows or back rest as circumstances require, so that bodily relaxation may be obtained. He should remain in bed constantly except to the degree to which the chair treatment may seem preferable, according to the considerations just discussed.

DIGITALIS

The mainstay of drug therapy in heart failure is digitalis. Despite the enormous amount of labor spent in the study of the pharmacologic action of digitalis, controversies still exist concerning its exact effects on the heart and peripheral circulation in normal and pathologic conditions. Its clinical value in heart failure needs no further proof. For practical purposes it matters but little nowadays what common preparation is used. There may be minor differences in potency but at present practically all digitalis in general use in this country has been satisfactorily standardized. There no longer is any need to purchase expensive preparations. The simplest method is to use the drug in the form of a pill, tablet or capsule. Equally good results can be obtained with a tincture, but it is less convenient to the patient and more difficult for the physician to know exactly how much is being taken. A drop is not a minim and the number of drops that equal one cubic centimeter will depend on the size of the dropper, the angle at which the dropper is held and the speed with which the drops fall. For these reasons a pill containing 0.1 Gm. (1½ grains) is recommended.

The patient, assuming he had no previous digitalis, may be given one pill orally four or five times the first day. A similar amount may be given the second day. It is expected, in the average case, that from fifteen to twenty pills will be necessary to effect digitalization. Because patients vary in the amount they require, it is important after the second day to watch for desirable or harmful effects. If no significant results have been obtained, the same dose of five pills may be continued for the third and fourth days. If partial effects have been noted, the daily dose may be cut down gradually to three or two. After a therapeutic result has occurred it generally is advisable to administer a so-called maintenance dose of about one pill (0.1 Gm. or 1½ grains) each day, which may be kept up more or less indefinitely. The toxic effects that would necessitate temporary omission of the drug are nausea and vomiting (not ascribable to heart failure or causes other than the drug), yellow vision, diarrhea or certain irregularities of the heart. The main disturbance of the heart itself that indicates toxicity is bigeminy due to ventricular extrasystoles. Other rare disturbances are complete heart block, undue slowing of the heart and very rarely auricular fibrillation. When first or second degree heart block results from digitalis some caution must be exercised in giving large doses, but no harm has been produced.

It must be borne in mind that digitalis is indicated in congestive heart failure whether the blood pressure is high or low, whether the rhythm is regular or grossly irregular and whether the rate is rapid or slow. It is to be used in myocardial or in valvular disease, whether fever is or is not present and whether the basal metabolism is normal or elevated. The results to be expected may differ under varying circumstances but the indication for its use remains the same.

If the condition of the patient is more critical and a more prompt effect is desired, digitalis should be given intramuscularly. For this purpose ampules containing one cat unit (0.1 Gm., or 1½ grains) are available. If no digitalis had been given in the previous several days, 0.5 Gm. may be injected deep into the gluteal muscles in one dose. This amount or a slightly smaller dose can be repeated in several hours if the circumstances warrant it. In fact, on very rare occasions when minutes seem precious, 0.5 Gm. may be injected intravenously. Results can be obtained in from one to two hours from intramuscular injections and in from fifteen to thirty minutes from intravenous injections.

There are many instances in which nausea, abdominal distress and even vomiting occur in congestive heart failure. Often the question arises whether these symptoms are due to digitalis. When abdominal complications such as gallstones or mesenteric thrombosis can be satisfactorily eliminated from consideration, it will generally be found that the situation requires more

2. Harrison, T. R.: Personal communication to the author.

rather than less digitalis. Occasionally careful electrocardiographic study will be necessary to make this decision. When auricular fibrillation is present and the apex rate is rapid, one can be fairly certain that insufficient digitalis has been given, provided hyperthyroidism is not present. When the cardiac rhythm is regular the fact that the rate is rapid is no proof that the heart can stand more digitalis. In some cases whether more or less digitalis is needed can be ascertained only through trial and error, for the main point is whether the clinical condition improves with one type of regimen or another. When gastrointestinal symptoms are prominent they may interfere with the administration of digitalis by mouth. In that case it can be given in the form of tincture rectally, the dose being about the same as the ordinary oral dose. For this purpose 4 cc. of a tincture in from 50 to 100 cc. of water may be injected daily for several days. When a cardiac patient in failure has already received an unknown amount of digitalis the foregoing regimen may need to be modified with smaller doses. One will have to use greater care, watching for toxic effects.

DIET

In the ordinary case of heart failure the dietary problem is not very important. Often it proves helpful to give a Karell diet for a few days, which consists of a glass of milk four times a day. This may be followed by small quantities of a general house diet. When there has been a history of dietary deficiency, vitamins, especially thiamine hydrochloride, should be given. Salt should be reduced to a minimum. It has been customary to restrict fluids to about 1 liter (quart) a day but I think it better to be more generous with the fluid intake, especially for older persons. It is well to avoid thirst and dryness of the mouth. When there has been considerable loss of fluid through sweating or if there is a tendency to an increase in the nonprotein nitrogen in the blood, it may be necessary to force fluids for short periods of time.

There is too little to be gained by purgation to compensate for the discomfort and weakening effect of frequent bowel movements. A bowel movement obtained once daily or every other day is all that is necessary. Simple cathartics or enemas may be used for this purpose.

SEDATION

Whenever necessary, cardiac patients should be helped to obtain a comfortable night's sleep. Mild sedatives such as chloral hydrate, phenobarbital or related products may be sufficient. Often they are ineffective and prolonged experimentation with the barbiturates should not be carried out, for many patients require narcotics when breathlessness or pain is the cause of restless nights. A hypodermic of one-sixth or one-fourth grain (0.01 or 0.016 Gm.) of morphine is generally advisable. It is a common experience that a patient suffering from congestive heart failure who has had poor nights shows the first evidence of improvement following the administration of morphine.

DIURETICS

In considering drug therapy of congestive failure, diuretics come next in importance to digitalis. Various preparations are available for this purpose and one may have to experiment with one or another medication to find which is most suitable in any particular case. Theophylline or aminophylline from 2 to 5 grains (0.13 to 0.3 Gm.) three times a day may be given orally. Sometimes a large single dose of these drugs repeated at intervals of five or seven days may be useful. These preparations may produce nausea, but when a suitable dose is tolerated it can be continued for long periods. In recent years the mercury diuretics have proved to be the most effective means to get rid of edema. When the ordinary regimen of rest and digitalis has not established satisfactory compensation, preparations like mercupurin may be injected intravenously or deep into the gluteal muscles. Care in injection is imperative, because distressing sloughs may result if some of the drug leaks out of the vein or is injected subcutaneously. The dose varies from 0.5 to 2 cc. and can be repeated every five to seven days, even for long periods. It is rarely necessary to give more than 1 cc. and then only when the smaller dose has begun to lose its effectiveness and no harmful results have occurred. Occasionally mercury suppositories may be effective but their use is limited because local rectal irritation often results and the diuretic effects are not as good as with the hypodermic method of administration. Mercury diuretics should not be given if acute nephritis is present. When a cardiac patient also has chronic nephritis, although one would prefer to avoid these preparations, at times they may still be useful. In older patients and in some younger ones who are very sick, occasionally ill effects from mercury diuretics occur. I suspect that we can minimize this danger by permitting a larger intake of fluid in these cases.

As a part of the administration of mercury diuretics it has been found that giving ammonium chloride (from 15 to 30 grains, or 1 to 2 Gm., three times a day) may improve the results. This may be continued in some cases for long periods. In fact, a diuresis on ammonium chloride may occur before the mercury is injected and occasionally it will be found that the patient becomes entirely edema free on this regimen alone and does not require a mercury diuretic.

MECHANICAL METHODS OF TREATMENT

Apart from the preceding outline of treatment for congestive heart failure there are other procedures that are required in individual cases. The physician should watch for evidence of hydrothorax, for if there is a significant amount of fluid in the pleural cavities (especially the right) thoracentesis will be necessary. Similarly if there is an embarrassing amount of ascites abdominal paracentesis should be carried out. In some cases effective diuresis by the use of the drugs discussed will be possible only after the removal of the abdominal fluid and the release of pressure on the renal vessels. Occasionally a distended bladder which prevents a diuresis from occurring is overlooked and needs catheterization. In a rare case of stubborn massive edema of the legs, Southey tubes may be employed. Finally the use of phlebotomy must be considered. We know that there is almost always a marked increase in blood volume in congestive failure. In cases in which there are marked venous distention and engorged liver, removing from 400 to 500 cc. of blood is often of great value. In cases of acute pulmonary edema or paraxysmal dyspnea, if improvement does not occur following a hypodermic injection of morphine, phlebotomy often gives prompt relief. A measure not quite as effective but with a similar purpose is to apply tourniquets around the four extremities, pooling the blood temporarily in the periphery.

It is obvious that the choice of these therapeutic measures will vary under different circumstances. In the mild cases of heart failure simple measures will be sufficient while in the very severe all procedures may need to be applied to attain compensation. In some

of the latter oxygen therapy, both for acute emergencies and occasionally for long periods, may prove valuable. When satisfactory clinical improvement has occurred or when a stationary condition has been reached, the patient may be gradually allowed to get out of bed and to increase his activities. In the process of rehabilitation it must be emphasized that the patient's subsequent life will need revision. The fact that heart failure developed (unless brought on by a specific cause such as infection) is evidence that the heart cannot tolerate the previous activities, and lessening of the burdens, both physical and emotional, is desirable. Longer rest periods are therefore necessary. In planning the details of these changes, cooperation between the family, the patient and the physician will be required.

PREVENTIVE AND CURATIVE MEASURES

The physician often has the opportunity to anticipate and thereby prevent or delay the development of heart failure in patients who have compensated heart disease. In this regard he must bear in mind the important precipitating or aggravating causes that tend to bring on decompensation. These are infections, overexertion, pregnancy, anemia, prolonged emotional strain, obesity, hyperthyroidism or any factor that significantly increased the work of the heart. As far as possible these should be avoided or minimized.

Finally, there are certain conditions in which heart failure is due to curable disease and the processes are reversible. Many instances of heart failure accompanying thyrotoxicosis can be dramatically relieved by subtotal thyroidectomy. A rare case of failure due to arteriovenous fistula can be cured by closing the shunt surgically. Heart failure due to myxedema, anemia or beriberi may respond readily to appropriate specific therapy. Heart failure during toxemia of pregnancy or with acute nephritis may disappear entirely under digitalis and other cardiac therapy. The physician should constantly be on the alert to discover the rare case of constrictive pericarditis, for this may be cured or greatly relieved by surgical measures and will not be helped at all by medical therapy. Infections may be the cause of heart failure and measures should be employed as far as possible to prevent infection. Furthermore, there are many cases in which heart failure is directly due to an abnormal rapid cardiac mechanism such as auricular tachycardia, flutter or fibrillation or to ventricular tachycardia. These almost always can be controlled by measures that stimulate the vagus nerve or by appropriate digitalis or quinidine therapy. It is clear that although the individual types of "curable heart failure" mentioned are not common, the entire group is not inconsiderable. The fact that they are curable lends great importance to these cases and should emphasize the need for accurate diagnosis.

SUMMARY

Among the various elements in the treatment of congestive heart failure rest in bed has always been regarded as most important. There are both theoretical and practical considerations which indicate that bed care may be actually harmful in certain cases and that rest in a chair is preferable. It is suggested that this question be reanalyzed in the light of newly acquired knowledge.

There is a small group of cases with congestive heart failure in which the condition is actually curable and the morbid processes reversible. Careful diagnosis is imperative if we are to recognize these cases.

270 Commonwealth Avenue.

Council on Physical Therapy

THE COUNCIL ON PHYSICAL THERAPY HAS AUTHORIZED PUBLICATION OF THE FOLLOWING CHAPTER, WHICH IS THE FIRST OF A SERIES ON AMPUTATIONS AND ARTIFICIAL LIMBS TO APPEAR IN THIS COLUMN. WHEN COMPLETED, THIS SERIES WILL BE PUBLISHED IN THE FORM OF A HANDBOOK ON AMPUTATIONS. THE COUNCIL WISHES TO EXPRESS ITS APPRECIATION FOR THE COOPERATION OF ITS GROUP OF CONSULTANTS ON ARTIFICIAL LIMBS. THE COUNCIL IS REPRESENTED BY DRS. FRANK D. DICKSON, HARRY E. MOCK, FRANK R. OBER, S. PERRY ROGERS, PAUL STEELE AND PHILIP WILSON, AND THE ASSOCIATION OF LIMB MANUFACTURERS OF AMERICA IS REPRESENTED BY MESSRS. MCCARTHY HANGER SR., W. E. ISLE, JOSEPH A. SPIEVAK, DAVID E. STOLPE AND J. B. KORRADY.

HOWARD A. CARTER, Secretary.

CHAPTER I. PSYCHOLOGIC AND PHYSIOLOGIC PRINCIPLES IN AMPUTATIONS

1. PSYCHOLOGIC FACTORS

The emotional reactions of patients subjected to amputation fall into three general groups, which correspond to the three main surgical indications for the operation.

The first group embraces those in whom an amputation is performed as an immediate result of an accidental injury. The patient, who has been previously well, active and physically whole, is suddenly confronted by the prospect of going through life maimed. His emotional shock is increased by the uncertainty of this prospect, by his ignorance of the amount of functional disability and disfigurement which his amputation will entail. The rapidity and completeness with which he adjusts himself to this situation will depend largely on his inherent emotional stability, but he can be assisted greatly in this adjustment if his physician is aware of the nature of his problem. The physician can be useful only to the extent that he is prepared to be explicit about just what the patient may expect to do with his stump and a good prosthesis. Emotional equilibrium is regained with a definite assurance of satisfactory appearance and function. As with all physically handicapped persons, harm is done by an excess of sympathy and coddling. The patient will regain his self respect and self reliance more rapidly if such an attitude is apparently expected by those about him.

The second group of patients embraces all those in whom amputation is performed because of chronic disease in the extremity, usually malignant tumor, chronic bone infection or vascular insufficiency. The major surgical problem in these cases is usually the saving of life; the creation of a functional stump and the use of a prosthesis are of secondary consideration. The patient is frequently seriously ill, feeble or debilitated. He is more or less resigned to ill health and incapacity. The loss of a physical member is not such an emotional shock as in the previous group. If his spirit is not completely broken he is likely to welcome any sort of prosthesis, however slight its esthetic or functional value, as a blessed improvement over his former state. However, even in amputations performed as life-saving emergencies, the foresighted surgeon can often create a useful stump at no additional surgical risk. The psychologic advantage to the patient should be sufficient incentive to such forethought whenever possible.

The third great group, and one which should possibly be larger, comprises those patients in whom amputation is performed in a deliberate effort to substitute a useful prosthesis for a useless, unsightly or hopelessly deformed extremity. The psychology of these patients is usually the antithesis of those in the first group. The

patient is frequently aware of the esthetic and functional possibilities and demands the amputation. The surgeon becomes the arbiter in this situation, and his judgment can be sound only if he is thoroughly familiar with the physiologic requirements for successful amputation stumps at all levels and if he is reasonably well acquainted with the types of prosthesis available. One patient must be persuaded to give up a part of his natural body, and the enthusiasm of another must be tempered by a credible explanation of the limitations imposed by his particular deformity. Once such a patient and his surgeon agree on amputation, however, the emotional attitude of the patient is always most favorable. Fortunately for the surgeon, such operations of election can be carefully planned, the leg-maker can be consulted, and the best functional type of amputation should therefore be obtained in every case. The personality change and the physical and spiritual regeneration of the patient delivered from a useless and disfiguring extremity and started afresh with a good prosthesis are among the most gratifying phenomena a surgeon is privileged to observe.

Patients from all three classes tend to have certain reactions in common as time goes on. Once the emotional adjustment to their physical loss is made, they settle down to make the most of life with a prosthesis. The shorter the period between amputation and prosthesis, incidentally, the easier the adjustment is likely to be. During the period of waiting and during the selection and manufacture of the prosthesis, the interest of the patient is likely to be centered on appearance. A shapely limb and a flesh-colored finish seem very attractive. Only after weeks or months of trial do the number and disposition of controlling straps, the shape and character of the socket, and the weight and balance of the prosthesis become important. Gratitude to the surgeon who has planned intelligently for function then begins to stir. The emotionally balanced patient always swings from the question of "How do I look?" to "What can I do?" If his stump and prosthesis are both designed for function, he does very well. He rapidly ceases to regard himself as abnormal in any way, he engages in as many physical activities as his individual disability will permit, and his family and friends soon forget that he is maimed.

2. PHYSIOLOGIC FACTORS

The physiologic functions of the upper extremity are complex. The finely coordinated movements of grasping, pushing, pulling and turning are exceedingly difficult to duplicate in mechanical apparatus. A gloved nonmechanical hand, made of wood, rubber or felt, may be worn for appearance only. Several types of mechanical hands are available in which the fingers or thumb, or both, are actuated by cords from a shoulder harness. The harness is usually arranged so that simple extension of the stump within its socket opens the fingers, which are closed by spring tension, and locked in different positions by an automatic ratchet. Various types of hooks, pliers and tool holders designed for manual labor are actuated in the same manner. Such apparatus requires of the stump only that it be actively movable, long enough to be grasped, and insensitive enough to exert leverage. Duplication of the more complex functions is approached only on the cineplastic amputations, in which skin-lined tunnels through the contractile bellies of antagonistic muscles are used to control complex motions of a mechanical hand. However, the function of cineplastic amputations is limited by the strength of the muscles available, by the durability of the tunnels and by the mechanical difficulties in manufacturing and fitting the appliances.

Substitution in the lower extremity is more successful because its functions are less complex. Its primary function is weight bearing. The requirements for a successful carrying out of this function in a stump may be inferred from an examination of the anatomy of parts of the body normally subjected to weight bearing. The heel, the front of the knee, the ischial tuberosity and the olecranon of the ulna are examples. The structure of the bone at these points is similar to that beneath the articular surfaces of joints, which are likewise pressure bearing areas. Such bone is always of the finely trabeculated cancellous variety, surrounded by only a thin cortex. Pressure is nowhere borne on tubular bone with its heavy cortex and open medullary canal. Pressure bearing bone is covered with either articular cartilage or a thin, adherent periosteum. The tendinous origins and insertions of muscles are found at most of these locations, and they withstand outside pressure well. The bellies of muscles are never subjected to pressure under normal conditions. If fascia is present it is adherent, never gliding. A minimum of areolar tissue and little or no fat are present. Even the skin over such points has characteristics in common. Both corium and epidermis are thicker, sebaceous glands are more scarce and sudoriferous glands are more plentiful. Furthermore, the specialized character of the skin in weight bearing areas is a congenital affair. Skin from other surface areas when subjected to pressure undergoes a certain amount of adaptation but never a true metamorphosis. Finally, skin subjected to pressure is not subjected to tension; the tension of muscular contractions and bone growth is taken up by the attachment of fascia and tendons to bone.

The second major function of the lower extremity is locomotion. Locomotion in an amputation stump depends on the mobility and the stability of the remaining joints and on the integrity of the neuromuscular apparatus. A muscle can exert no dynamic function in a stump unless, first, its normal length is preserved, second, its origin and insertion are both attached to bone, and, third, its course crosses a movable joint. The death of muscle left without function is a prominent cause of atrophy in an amputation stump.

Both major functions of the lower extremity are dependent on the relative absence of pain. Pain is frequently the major cause of limp. Any patient of normal mentality can learn to walk gracefully on a stump which he can move and on which he can bear weight without pain. But pain from external pressure, pain within the stump from any cause, or the pain of fatigue preclude satisfactory function following any amputation.

Certain mechanical factors are also fundamental. Weight bearing means pressure on a horizontal body surface; that is, one at right angles to the force of gravity. An oblique surface is capable of weight bearing only as its plane approaches the horizontal. As its plane approaches the vertical, the amount of surface pressure required to exert one unit of downward force increases according to geometric law. A corollary to this proposition is that the larger the horizontal surface the less pressure per square unit of area is required to support a given weight. The mechanical requirements for locomotion are just as simple. Motion of a stump can be transmitted to a prosthesis only if effective leverage is available through surfaces that can withstand lateral pressure. And, finally, locomotion requires that the prosthesis be lifted and turned as well as propelled.

Council on Pharmacy and Chemistry

PRELIMINARY REPORT OF THE COUNCIL

THE COUNCIL HAS AUTHORIZED PUBLICATION OF THE FOLLOWING PRELIMINARY REPORT.

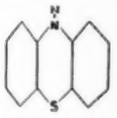
BECAUSE OF RECENT INTEREST SHOWN IN THE EXPERIMENTAL WORK ON PHENOTHIAZINE AS A PARASITICIDE AND AS AN ANTIBACTERIAL AGENT, IT IS EXPECTED THAT THE MEDICAL PROFESSION MAY SHORTLY NEED MORE INFORMATION ON PHENOTHIAZINE OR ITS DERIVATIVES. THERE HAS ALREADY BEEN PUBLISHED IN THE JOURNAL A REPORT ON A SERIES OF CASES OF DERMATITIS FOLLOWING EXPOSURE TO PHENOTHIAZINE AS AN AGRICULTURAL SPRAY. ACCORDINGLY, A PRELIMINARY REPORT ON THE PRESENT STATUS OF PHENOTHIAZINE HAS BEEN PREPARED. THIS PREPARATION IS IN THE EXPERIMENTAL STAGE FOR HUMAN USE.

PAUL NICHOLAS LEECH, Secretary.

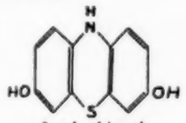
PHENOTHIAZINE

Phenothiazine is the parent substance of a large number of dyes, three of which are the thiazine dyes thionol, Lauthe's violet and methylene blue. In 1935 Smith and his co-workers [1] reported an original piece of research on the value of phenothiazine as an insecticide. A substance that is of high toxicity to insects and of low toxicity to warm-blooded animals has long been sought by the U. S. Department of Agriculture [2] and it was believed that a promising substance had been found in this new agent. Further work led to a number of other practical uses, one of the most important being the treatment of some worm infestations of cattle, sheep, swine and horses and the control of mosquito larvae in ponds. More recent work [3] has revealed the substance to be effective in the treatment of certain cases of urinary tract infections.

Phenothiazine is prepared from diphenylamine and sulfur. Phenothiazine, thionol and leukothionol (two excretion products) have the following structural formulas respectively:


Phenothiazine.

Thionol.


Leukothionol.

Phenothiazine is distantly related to sulfanilamide. It is a fine pale yellow powder with practically no taste, and it is almost insoluble in water. Exposure to air and moisture causes a slow spontaneous oxidation in which lies its value as both an insecticide and a fungicide. In its original form phenothiazine is effective as an insecticide, but in oxidation it is changed to phenothiazone, in which form it is active as a fungicide.[3] The amount present in apple-spray residues may be estimated by a colorimetric method [4] which is based on the conversion of phenothiazine to a highly colored red compound.

The effectiveness of phenothiazine as a urinary bactericidal agent was discovered when it was noticed that the urine of animals receiving the drug turned red on exposure to air and that on standing for considerable time the urine did not develop the usual disagreeable odor.[5] Subsequent examinations revealed lack of gross evidence of bacterial growth in the urine [6] after standing exposed for several weeks.

Preliminary tests were then made of the urine of phenothiazine fed rats on cultures of Escherichia coli. Sufficient retardation of growth occurred to warrant further investigation in rabbits.[6] No toxic effects were observed and one of the authors administered the drug to himself and collected several specimens of urine at varying intervals of time. The urine again was shown to have an antiseptic action on Escherichia coli. Experimental cystitis was produced in male rabbits, phenothiazine given, and its effect noted on the infection. Bacteriologic examination of the urine resulted in sufficient encouragement to warrant further trials by increasing the acidity of the urine and conducting tests on human beings known to have urinary tract infections.[3]

In the treatment of thirty-one patients with chronic and sixteen with acute infections of the urinary tract, DeEds, Stockton and Thomas [3] used phenothiazine in average daily doses of 1.33 Gm. for an average of 7.4 days. Ten patients with infections were permanently relieved of symptoms, and pus and bacteria disappeared from the urine. Cystoscopic examination revealed an increased bladder capacity and decreased hyperemia. Five patients with chronic inflammation were cured and twenty were improved. Only eight of the forty-nine patients failed to secure relief from symptoms if the urine was kept sufficiently acid (p_H 4.5 to 5.5). No undesirable effects could be demonstrated in the gastrointestinal tract, circulation, kidneys or liver. Anemia occurred in three cases after an average total dose of 23.3 Gm., which is far beyond the usual therapeutic dose. The anemia was apparently hemolytic in nature, since there was an early reticulocyte response and a rapid regeneration of the blood. Previous experiments on albino rats [5] had shown that continued feeding fails to cause any demonstrable tissue injury except in very large doses in which retardation of body growth occurred. The bactericidal properties of phenothiazine are due [6] to its being excreted in urine partly as the original compound, partly as thionol and its leuko base, and partly as leuko thionol in loose chemical combination. On hydrolysis in sufficiently acid urine free leuko thionol is obtained [7] which in turn is oxidized to thionol, the true antibacterial agent.

The possibility of the value of phenothiazine as an agent to combat human trichinosis was evolved with the report [8] that its continuous use over a period of six weeks reduced the severity of trichinosis in rats by 74 per cent. However, a later report [9] by the same authors asserted that no evidence was found to show phenothiazine to be of any value in experimental trichinosis in rats.

More recently a communication appeared in THE JOURNAL [10] describing patients complaining of skin irritation following the use of phenothiazine while using the material as a spray. Because of failure to obtain similar reactions by men working in laboratories, an investigation was made. It was found that the reactions were due to the excretion of the reversible oxidation-reduction system thionol-leukothionol previously reported.[11] Leukothionol is photosensitive and is oxidized to thionol when exposed to sunlight under anaerobic conditions. It was suggested, then, that the reactions were due to photosensitization and not to local irritation by phenothiazine.

It is understood that a number of pharmaceutic houses have distributed phenothiazine for investigational use. The product appears to have unusual properties both as a urinary bactericidal agent and as a possible treatment for worm infestations. Although the product is still in the experimental stage as regards therapeutic use, because of these unusual properties it is almost certain to obtain much attention in the near future. It is of more than academic interest, in that the material is also used as a spray in agricultural industry and has been shown to be the cause of cutaneous reactions.

The preparation is one which has interesting possibilities; there is lacking sufficient evidence both from the chemical point of view and from that of clinical research to justify general use by the medical profession at this time. In view of the experimental nature of the drug the Council suggests that further investigations with adequate controls will be useful for a proper evaluation of the therapeutic possibilities of phenothiazine.

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THE JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION

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SATURDAY, NOVEMBER 16, 1940

THE PLATFORM OF THE AMERICAN MEDICAL ASSOCIATION

The American Medical Association advocates:

1. The establishment of an agency of the federal government under which shall be coordinated and administered all medical and health functions of the federal government exclusive of those of the Army and Navy.

2. The allotment of such funds as the Congress may make available to any state in actual need, for the prevention of disease, the promotion of health and the care of the sick on proof of such need.

3. The principle that the care of the public health and the provision of medical service to the sick is primarily a local responsibility.

4. The development of a mechanism for meeting the needs of expansion of preventive medical services with local determination of needs and local control of administration.

5. The extension of medical care for the indigent and the medically indigent with local determination of needs and local control of administration.

6. In the extension of medical services to all the people, the utmost utilization of qualified medical and hospital facilities already established.

7. The continued development of the private practice of medicine, subject to such changes as may be necessary to maintain the quality of medical services and to increase their availability.

8. Expansion of public health and medical services consistent with the American system of democracy.

PUBLIC HEALTH AND THE MEDICAL PROFESSION

Public health in the United States has been one of the most important contributions of organized medicine to the public welfare. When the American Medical Association was organized in 1847 there were no state health departments and few city health departments. In many instances "the local medical societies took an active interest in instituting measures for the protection of their communities against pestilential disease and in some localities attended to this work without any form of health organization" (Rise and Fall of Disease in Illinois).

At the first meeting of the American Medical Association in 1847, matters pertaining to hygiene and sanitation were leading subjects. Even the preliminary meeting in 1846 urged "upon the several state governments the adoption of measures for a registration of births, marriages and deaths." Such vital statistics are an important feature of every modern health department. In 1848 the Committee on Public Hygiene of the American Medical Association expressed the hope, in its first report, "that the day is not far distant when a town without sewerage will be unknown in the United States." At the Association's meeting in 1849, it was emphasized, "pure water, like pure air, is so essential a requisite that its presence is absolutely necessary to preserve human life." The abatement of nuisances was also discussed at this meeting, for "the necessity for the removal of the refuse which is constantly accumulating in streets is . . . obvious." Present day health departments employ competent sanitary engineers for the control of these problems.

Epidemiology and communicable disease control were not neglected. In 1853, in the absence of health departments, it was "resolved, that the Association earnestly recommend to the local societies in different portions of our country to appoint committees, whose duties it shall be to record the prevalence of epidemics or other diseases." The next year it is found that the delegates "demand the speedy enactment of further and more stringent quarantine laws to prevent the spread of infectious disease."

These few citations from the records of the American Medical Association could be multiplied many times, showing the attitude of the medical profession to public health. They served to stimulate physicians in the different states to form state health departments. The first state health department to be established was that of Massachusetts, organized in 1869 after thirty years of endeavor by the medical profession. In 1861 it was

> *Resolved*, That the Massachusetts Medical Society petition the Legislature to grant the petition . . . for a State Board of Health . . .
>
> That every member of the Medical Society be requested to use his influence . . . to support this measure in the Legislature.

California in 1870 was the second state to establish a state board of health, under the guidance of leaders in the state medical society. The Medical Society of Virginia, reorganized in 1871 after the Civil War, at its first meeting petitioned the state legislature to establish a state board of health, which was done in that year. Thus the record goes for state after state. In Alabama the state medical association agitated the problem of a state board of health for several years, until in 1875 the Alabama general assembly passed a law designating the Alabama State Medical Association as the state board of health and county medical societies affiliated with the association as boards of health in their respective counties. In 1874 the Tennessee State Medical Society prepared and presented to the legislature a bill providing for the establishment of a state board of health. In 1875 this failed to pass; in 1877 it was passed but the appropriation was stricken out. In 1876 the Illinois State Medical Society felt that "a board of health organized and provided with the means to collect information, and diffuse among the people

a better knowledge of the laws of health, and to discover to them the dangers to which they are exposed, would save thousands of poor victims from untimely death."

Scarcely any state health departments and few city health departments exist that do not owe their origin to the activities of state and local medical societies. The years have not decreased the active interest of medicine in public health work. True, honest differences of opinion have arisen at times in the administration of public health activities (as they have arisen in other government functions); unfortunately some self-promoting opportunists have seized these occasions to make it appear that medical organizations are opposed to public health activities.

Among the officers, the Board of Trustees and members of the House of Delegates of the American Medical Association are members of state and city boards of health. In these capacities they have been active in helping to frame the policies of the Association. Even as lately as 1938 the House of Delegates of the American Medical Association at a special session emphasized the desirability of "the strengthening and expansion of recognized public health service in states and local communities." Here is no opposition or enmity for public health. The honest differences of opinion which do exist have been centered definitely on the extent to which public health officialdom shall depart from its primary function of prevention of disease and take over the practice of medicine.

SULFANILAMIDE IN THE LOCAL TREATMENT OF WOUNDS

The precise mechanism of the specific chemotherapeutic action of sulfanilamide on bacteria has not been conclusively elucidated. Colbrook, Bliss and Long, Lockwood and others demonstrated that the drug has a bacteriostatic action in vitro on hemolytic streptococci, staphylococci, pneumococci and colon bacilli. The extent of this action, however, as pointed out by Fleming and by Lockwood and Lynch, depends on the concentration of the drug and on the concentration of peptones in the culture medium. The action of the drug in a wound may be modified by a number of factors. Destruction of tissue results in the breakdown of protein with formation of peptone, and the presence of peptone inhibits the bacteriostatic action of sulfanilamide.

Jensen, Johnsrud and Nelson [1] produced experimental open fractures of ribs in a series of twenty-seven guinea pigs. The wounds were flooded with a culture of a virulent strain of Staphylococcus aureus and were sutured. In a control series 71.4 per cent became infected, while in the series in which 0.5 Gm. of sulfanilamide was placed in the wound only 20 per cent became infected. In a series in which 0.5 Gm. of sulfanilamide was placed beneath the skin of the abdomen 80 per cent became infected, demonstrating the inferiority of the systemic action of sulfanilamide. At the Minneapolis General Hospital the same authors treated thirty-nine compound fractures and two compound dislocations by placing crystalline sulfanilamide into the wounds after completion of meticulous débridement. The wounds were closed by interrupted silk sutures to the skin only. Complete reduction of the fracture was accomplished at the time of débridement, and absolute immobility by immediate application of unpadded plaster splints. All of the wounds healed by primary intention. Ninety-four compound fractures treated at the same hospital by similar methods, except that sulfanilamide was not used, showed 27 per cent infection, seven cases of gangrene and five amputations to control infection. The authors explain these truly striking results by the fact that, while systemic administration of sulfanilamide aims to produce a concentration of from 10 to 15 mg. per hundred cubic centimeters in the blood and tissue fluids, the local implantation produces a concentration of about 800 mg. per hundred cubic centimeters in the contaminated area and maintains this concentration over a period of about thirty-six hours. Thus the local implantation of sulfanilamide appears to be a highly efficient method of preventing and combating infection in compound fractures. The method is rapidly becoming the most important addition to the treatment of wounds of the type most frequently met in industrial surgery and in military surgery and is being rapidly adopted in surgical practice here and in England. Key and Burford [2] investigated in animal experiments the question of whether or not the local implantation of sulfanilamide in a compound fracture inhibits healing of the bones. They produced bilateral symmetrical fractures in one bone of each foreleg in a series of fifteen rabbits. The wound in the right foreleg was packed with crystals of sulfanilamide; the fracture in the left foreleg served as a control. Roentgenographic and microscopic studies failed to reveal any differences in the formation of callus on the two sides. From their clinical as well as experimental observations they conclude that the local implantation of sulfanilamide crystals in compound fractures not only tends to lessen the danger of infection but also does not perceptibly interfere with the union of the soft tissues or of the bone.

Mayo and Miller [3] report on the local use of sulfanilamide in cases after combined abdominoperineal or posterior resections for carcinoma of the rectum. The drug was applied directly to the wound, either by packs saturated with the solution or by direct irrigation of the wound. The solution was prepared by adding sulfanilamide to boiling physiologic solution of sodium chloride (0.8 per cent is the limit of solubility of sulfanilamide). Irrigations with this chemical compound

1. Jensen, N. K.; Johnsrud, Luverne W., and Nelson, M. C.: The Local Implantation of Sulfanilamide in Compound Fractures, Surgery 6:1 (July) 1939.

2. Key, J. A., and Burford, T. H.: The Local Implantation of Sulfanilamide in Compound Fractures, South. M. J. **33**: 449 (May) 1940.

3. Mayo, C. W., and Miller, J. M.: Solution of Sulfanilamide in the Local Treatment of Wounds, Proc. Staff Meet., Mayo Clin. **15**: 609 (Sept. 25) 1940.

have been effective when other drugs have failed. The wounds were much cleaner in appearance and presented healthy granulation tissue. Herrell and Brown [4] of the same clinic used a thick suspension by adding 2 Gm. of sulfanilamide to 100 cc. of an 0.8 per cent solution of sulfanilamide in physiologic solution of sodium chloride in the treatment of twenty-one cases of badly infected wounds of the scalp, thorax and pericardium, abdominal incisions and infected sinuses. The results, in their opinion, have been sufficiently encouraging to justify a continuation of this procedure.

"JUG STATION" MILK

In 1932 THE JOURNAL condemned the sale and use of bootleg milk,[1] namely milk offered for sale outside municipal limits and therefore not subject to the regulations governing the sale of milk within the municipality. Several health officers at that time were confronted with the health hazard developed by the sale of milk at roadside stands to residents of nearby cities. Such milk was cheaper than milk sold in the city. Often it came from farms of such insanitary condition that their product could not be offered within the city limits.

Studies by the United States Public Health Service and the former American Child Health Association have demonstrated again and again that milk-borne epidemics are due almost exclusively to raw milk supplies.[2] In many parts of this country, especially the smaller cities, raw milk still constitutes an appreciable percentage of the daily milk distribution. Raw milk unless certified is frequently produced under conditions not conducive to safety. The danger of contamination of raw milk has been recognized even to the extent that producers of certified milk and the American Association of Medical Milk Commissions, which supervises certified milk production, are agreed that pasteurization would be an additional safeguard even for certified milk.[3]

The sale of raw milk continues in spite of the fact that pasteurized milk is safer. Certainly there is no established evidence to indicate that raw milk is any more desirable nutritionally than pasteurized milk. The Council on Foods and Nutrition of the American Medical Association has published the following decision concerning the relative nutritional values of raw and of pasteurized milk:

> Milk is an excellent medium for many dangerous bacteria as well as an excellent food for man. Disease germs may enter the milk directly from an ailing cow, be introduced by insects, or be transferred to the milk by the fingers or mouthspray of persons having to do with the collection or transportation of milk. . . .
>
> The pasteurization of milk is a public health measure. The public should demand pasteurized milk for drinking and the use of pasteurized milk in milk products. The dairy trade should universally adopt pasteurization in the interest of public health.
>
> There is no cogent evidence that pasteurized milk is significantly inferior nutritionally to raw milk.

Even today bootleg milk stations, also called "jug stations," still operate outside the corporate limits and consequently outside the jurisdiction of some cities in which only pasteurized milk, other than certified, is legal. When every possible legal precaution has been taken to protect a community against potential dangers in raw milk, and milk consumers persist in circumventing the local regulations by buying milk from unsupervised sources, the occurrence of milk-borne outbreaks of communicable disease cannot be attributed to any failure on the part of the health authorities. Unless county and state authorities cooperate with city health officials in controlling this menace to health, outbreaks of milk-borne infection are bound to occur.

Current Comment

ORGANIZATION OF MEDICINE AND MILITARY SERVICES

As our nation begins to mobilize for efficient defense against any type of aggression, the medical profession is called on increasingly for the specialized service which only medicine can render. The pages of THE JOURNAL have been reflecting these activities increasingly in recent weeks. It is trite to say that medicine has advanced more greatly in the past fifty years than in all the previous centuries of the life of man. It has, moreover, become even more specialized in the twenty years that have passed since the last war than in the twenty years previously. The increasing specialization in medical service has introduced into medical practice innumerable technicians of considerable variety. The technical branches associated with medical care include physical therapists, roentgenologists, clinical pathology technicians, lay anesthetists and dental hygienists, as well as the vast personnel of the hospital, including the record librarians, the dietitians, the hospital librarians, makers of splints and braces, and other similar workers. Moreover, there are vocations associated with medicine which do not involve complete medical training but which have in many places legal recognition. Thus there are masseurs who are essentially physical trainers or rubbers rather than physical therapists. There are podiatrists who in some instances, under medical supervision, are helpful in the treatment of lesions of the soft tissues of the feet. In addition there are osteopaths, chiropractors, naturopaths and others who claim to have training and ability to treat human diseases and abnormalities. In the evolution of the medical services associated with military establishments recognition has been sought by many of these agencies, and attempts have been made to develop suit-

4. Herrell, W. E., and Brown, A. E.: Local Use of Sulfamido Compounds in the Treatment of Infected Wounds, Proc. Staff Meet., Mayo Clin. **15**: 611 (Sept. 25) 1940.

1. Bootleg Milk, editorial, J. A. M. A. **99**: 1606 (Nov. 5) 1932.

2. Frank, L. C.; Clark, F. A.; Haskell, W. H.; Miller, M. M.; Moss, F. J., and Thomas, R. C.: Do Children Who Drink Raw Milk Thrive Better than Children Who Drink Heated Milk? Pub. Health Rep. **47**: 1941 (Sept. 23) 1932. Crumbine, S. J.: Report on Milk-Borne Epidemic Diseases in the United States and Canada, 1924 to 1933, Am. Child Health A. Bulletin, July 1934.

3. Pasteurization of Certified Milk, editorial, J. A. M. A. **105**: 601 Aug. 24, 1935.

able rank and advancement for workers in every one of these fields. Some of these movements have gone to the point of proposed legislation, and attempts have been made to secure official status for workers in some of these fields which would carry them not only to the ranks available in the lower grades of the medical corps, but even to lieutenancy, captaincy and colonelcy. In military organizations each rank provides a certain income with certain command or professional responsibilities. Physicians, dentists and veterinarians have rank commensurate with their responsibilities. It would be most unfortunate if members of a technical group that may be of assistance to the medical profession in a subordinate capacity should be advanced by law in military service to rank granting them not only equality but actual supervision over highly trained physicians and surgeons. Unfortunately, in their desire for advancement, several of those engaged in the technical branches that have been mentioned have urged the aid of physicians in their campaign for legislation. The doctors will do well to avoid such entangling alliances and avoid participation in such movements unless they are completely aware of what the ultimate effect might be on the medical profession, and particularly on that part of it in the military service.

LACK OF PROVITAMIN A IN FLOUR

A small amount of yellow coloring matter is naturally present in wheat. This pigment formerly was considered to be largely, if not entirely, carotene. Because the coloring material is completely destroyed by the bleaching process to which much white flour is subjected, the question naturally has arisen whether such bleaching does not materially impair the nutritive value of the flour because of the destruction of carotene, which is provitamin A. The older reports in the scientific literature have indicated that such might be the case. Drummond and Wilbraham,[1] in their essays on diet, have compared the carotene content of unbleached and bleached white flour. A review of the available evidence led these authors to conclude that unbleached flour may contain as much as 0.2 Gm. of carotene to each hundred grams, whereas the carotene content of bleached flour is nil. The Council on Foods and Nutrition has also reviewed the evidence on the alleged carotene content of flour.[2] The Council has calculated that a pound loaf of bread made from unbleached flour may furnish from 0.6 to 1.2 mg. of carotene, which would be equivalent to from 1,000 to 2,000 U. S. P. units of vitamin A or roughly about one half the daily requirements for an adult. These calculations, of course, were made on the assumption that the reported values of the carotene content of wheat and its various products are substantially correct. Not many bio-assays of wheat or its products have been reported, but these have shown the vitamin A value to be small. It now appears from a recent investigation by Zechmeister and Cholnoky [3] that the natural pigment of wheat largely consists of xanthophyll, a yellow coloring material related to carotene but having no vitamin A activity. Small quantities of crystalline xanthophyll have been isolated from flour. By means of chromatographic experiments these authors found that the unbleached flours which they examined are almost completely free of carotene. Because cryptoxanthine is also absent, they conclude that flour is valueless as a source of provitamin A. The determinations were made on Hungarian wheat flour. It would be well to repeat the experiments with the new technic on various milling fractions of wheat as well as grain from different localities. Until evidence to the contrary is obtained, however, it would be proper to consider both bleached and unbleached flour as meritorious foods but devoid of or insignificant as sources of vitamin A activity.

1. Drummond, J. C., and Wilbraham, Anne: The Englishman's Food, A History of Five Centuries of English Diet, London, Jonathan Cape, 1939.
2. Accepted Foods and Their Nutritional Significance, Council on Foods of the American Medical Association, 1939, p. 102.
3. Zechmeister, L., and Cholnoky, L.: Carotenoids of Hungarian Wheat Flour, J. Biol. Chem. **135**: 31 (Aug.) 1940.

ARMOR IN MODERN WARFARE

At the time of the last war many wounds and injuries from projectiles and bomb fragments could have been avoided with the use of suitable protective devices for the body—helmets and armor—and this is true perhaps to an even greater degree in the present war. Apparently most of the armed services in different countries have recognized this problem and are studying it and making experiments. The major technical considerations are the speed of the projectile against which protection is most necessary, the regions of the body most vulnerable and most vital and the weight and composition of the armored material. One of the principal students of this subject has been Mr. Kenneth Walker of London, who among his other contributions to this discussion maintains that by restricting protection to certain areas of the body it is possible to employ armor of a weight which will protect from missiles of even high velocity (2,600 feet per second—the velocity of a machine gun bullet fired at close range). The problems are complex but Mr. Walker has also pointed out that, if anything is achieved along the lines of better armored protection, the initiative must come from the medical profession.

BUILDING HOSPITALS

The impression prevails and in certain circles is widely promoted that hospital construction has halted during the depression. True, many of the sources from which income for hospital building is drawn in prosperous times have lessened their flow. However, the WPA in a "Report of Work Completed, 1935-1940" relates that during this period 132 new hospitals have been constructed, 1,592 have been reconstructed or improved and additions have been made to 222. Up to June 1939 the PWA in its report "America Builds," page 143, states that "PWA has provided 121,760 beds in hospitals costing $367,659,880." The technical committee which reported to the National Health Conference, and from which report much of the impression has proceeded, estimated that a normal increase in hospital beds should be 25,000 a year. Since the PWA alone provided 121,760 beds in five years, it should be evident that the alarm of the National Health Conference with regard to hospital construction is somewhat lacking in justification.

MEDICAL PREPAREDNESS

In this section of The Journal each week will appear official notices by the Committee on Medical Preparedness of the American Medical Association, announcements by the Surgeon Generals of the Army, Navy and Public Health Service, and other governmental agencies dealing with medical preparedness, and such other information and announcements as will be useful to the medical profession.

PHYSICAL REQUIREMENTS THE SAME FOR ALL WHO ENLIST OR ARE INDUCTED INTO ARMY

The War Department has announced that the same procedure in physical examination and the same standards of physical requirements prescribed for Class 1-A registrants will be used for the physical examination of (1) men enlisted or reenlisted in the Regular Army, (2) men enlisted or reenlisted in the National Guard, (3) enlisted men of the National Guard inducted into federal service, (4) men enlisted in the Army of the United States, (5) men inducted into the Army under the Selective Training and Service Act of 1940. Examinations will be conducted with the utmost care in order that no individual who is unfit for military service will be accepted. Examining physicians will consider the standards set forth in the regulations as a guide to their discretion rather than construe them too strictly or arbitrarily.

The standards in the new regulations apply only to applicants for enlistment or induction into the Army of the United States. Physical requirements for all other personnel (candidates for commission in the Army or the Air Corps, candidates for appointment as Army nurses, warrant officers and candidates for the United States Military Academy) are defined in other army regulations.

Examinees will be classified into three categories in accordance with the conclusions of the medical officers conducting the physical examination:

Class 1-A: Those physically qualified for general active military service.

Class 1-B: Those who are physically unfit for general active military service but are fit for special and limited military service.

(Registrants who fail to qualify for Class 1-A and who do not fall below Class 1-B in any phase of the examination will be placed in Class 1-B unless because of multiple defects the medical examiners recommend unqualified rejection and placement in Class 4. Men placed in Class 1-B will not be accepted unless specific directions to that effect are issued by the War Department.)

Class 4: Those physically unfit for any military service. (All registrants who do not meet the physical requirements of Class 1-A or Class 1-B will be placed in Class 4.)

No applicant will be accepted for enlistment or induction who does not meet the standards prescribed for vision and hearing or who suffers from toxic conditions associated with abnormal conditions of the ductless glands, from valvular disease of the heart, from tuberculosis, from communicable diseases, from mental disease or deficiency, from insufficient teeth or from irremediable defects of the feet. Medical examiners will reject all men in need of hospitalization and all those who by reason of physical defects are considered unfit for early participation in training activities.

Whenever it appears to an examining physician that a registrant is endeavoring to escape military service by malingering, if he is otherwise mentally and physically fit the registrant will be accepted for military service.

PHYSICAL STANDARDS UNDER SELECTIVE SERVICE

On October 18 the physical standards under the Selective Service, which were published in THE JOURNAL November 2, were modified by a special circular issued by the chief of staff which has just become available. According to this special circular, which modifies physical standards not only for men selected under the Selective Service but for enlisted men as well, the presence of malaria, uncinariasis and remediable incapacity due to recent acute illness or injury or to employment or environment in civil life will not be considered as permitting the man to be placed in Class 1-A and as suitable for military service. Moreover, the section on "Genito-Urinary Organs and Venereal Diseases," which appeared on page 1557, is to be modified by the elimination of the classifications "Gonorrhea, acute or chronic," "Syphilis with remediable manifestations except cerebrospinal, cardiovascular or visceral syphilis" and "Chancroids and the resulting infections of the lymph glands of the groin." This, however, is still further modified by a telegram which was received last week from Mr. Dykstra indicating that two positive Wassermann tests or any venereal disease would be considered as causes for rejection.

Under the heading "Endocrine and Metabolic Disorders," which appeared on page 1558, the heading under Class 1-A *e* is to be eliminated, i. e. pellagra, beriberi, scurvy and other nutritional deficiencies, if remediable by correction of diet and not severe, will no longer be considered as conditions which would qualify for general military service.

Finally, under the heading of "Diseases of the Blood and Blood-Forming Tissues," all the items listed under 1-A are eliminated so that no longer can secondary anemia, purpura, sickle cell anemia or malaria be considered as conditions which would qualify for general military service.

These deletions are explained in the circular No. 117, which was issued on October 18, in the following paragraph: "The defects noted in the deleted paragraphs will be regarded as causes for rejection by Army medical examiners until there has been recovery without disqualifying sequelae. Medical examiners will reject all men who are in need of hospitalization, and all those who by reason of physical defects are considered unfit for early participation in training activities."

This is in accord with the expressed statement that the purpose of the Selective Service is to secure men physically fit for military training at the time of examination and not primarily to rehabilitate those who are found to be unfit. No doubt, however, every encouragement will be given to those who are unfit to seek recovery from the conditions for which they are rejected so as to be available subsequently.

RESERVE OFFICERS ORDERED TO ACTIVE DUTY

ARMY—SECOND CORPS AREA

Following is a list of the medical reserve officers with grade, permanent address and military area, ordered to active duty by the commanding general, Second Corps Area, up to November 8:

LIPPOLD, William E., Lieut. Col., Brooklyn, 2d.
JOHNSON, Vansel S., Major, New York, 2d.
KASICH, Milosh, Major, New York, 2d.
LYON, Edward C., Major, New York, 2d.
PUGH, Walter S., Major, Utica, N. Y., 1st.
RUSSELL, Lecky H., Major, Elmhurst, L. I., N. Y., 2d.
YELLEN, Hiram S., Major, Buffalo, 1st.
BEERY, Edwin N., Captain, Brooklyn, 2d.
CANTOR, Mortimer J., Captain, Brooklyn, 2d.
DEUTCH, Theodore M., Captain, Brooklyn, 2d.
DISICK, Solomon, Captain, Brooklyn, 2d.
EASTMAN, Harry D., Captain, Albany, 1st.
EDELSTEIN, Isidore S., Captain, Brooklyn, 2d.
FEIGIN, Emanuel V., Captain, New York, 2d.
HERR, Bayard S., Captain, Amsterdam, N. Y., 1st.
KETTLER, George H., Captain, Menands, N. Y., 1st.
KERSTEIN, Louis N., Captain, Rochester, N. Y., 1st.
MAYER, David E., Captain, New York, 2d.
MICHELSON, Henry, Captain, Paterson, N. J., 3d.
NEUWIRTH, Abraham A., Captain, Floral Park, L. I., N. Y., 2d.
NICOLAIS, Michael A., Captain, Trenton, N. J., 3d.
REED, Theodore D., Captain, Amityville, L. I., N. Y., 2d.
ROSENBERG, Albert B., Captain, North Plainfield, N. J., 3d.
SABIN, Anthony G., Captain, Brooklyn, 2d.
SICILIANO, Thomas, Captain, Brooklyn, 2d.
STARK, Jesse D., Captain, New York, 2d.
YACHNIN, Samuel, Captain, Lyndhurst, N. J., 3d.
AMATO, Vincent, 1st Lieut., Astoria, L. I., N. Y., 2d.
BARBANO, Alfred J., 1st Lieut., New Brunswick, N. J., 3d.
BLICK, Michael S., 1st Lieut., New York, 2d.
BLOOM, Bernard, 1st Lieut., Brooklyn, 2d.
BRITT, Richard W., 1st Lieut., Tonawanda, N. Y., 1st.
BRUSSEL, James A., 1st Lieut., Brentwood, L. I., N. Y., 2d.
CHERNUS, Jack, 1st Lieut., Newark, N. J., 3d.
CONANT, Ralph E., 1st Lieut., Kew Gardens, L. I., N. Y., 2d.
CRANE, Timothy F., 1st Lieut., New York, 2d.
CREIGHTON, James J., 1st Lieut., Buffalo, 1st.
DAVOLOS, Joseph J., 1st Lieut., Wilmington, Del., 3d.
DINKIN, Sidney, 1st Lieut., Brooklyn, 2d.
DOBSON, Leslie H., 1st Lieut., Poughkeepsie, N. Y., 2d.
DONALD, Howard A., 1st Lieut., South Ozone Park, N. Y., 2d.
DURHAM, James R., 1st Lieut., Wilmington, Del., 3d.
EBY, Robert E., 1st Lieut., New Rochelle, N. Y., 2d.
FABRICANT, Harry, 1st Lieut., Brooklyn, 2d.
FEIGEN, Gerald M., 1st Lieut., Paterson, N. J., 3d.
GILBERT, Sidney B., 1st Lieut., New York, 2d.
GOLDSTEIN, Joseph D., 1st Lieut., Jersey City, N. J., 3d.
GOLDSTEIN, Solomon, 1st Lieut., Brooklyn, 2d.
GOODWIN, Albert F., 1st Lieut., Schenectady, N. Y., 1st.
GRANT, Raymond J., 1st Lieut., Jersey City, N. J., 3d.
GRAYSON, Morris, 1st Lieut., Ozone Park, L. I., N. Y., 2d.
GREENFIELD, Sylvan J., 1st Lieut., Newark, N. J., 3d.
GROOPMAN, John, 1st Lieut., New York, 2d.
GROSS, Charles N., 1st Lieut., Atlantic City, N. J., 3d.
GUIDOTTI, Frank P., 1st Lieut., Trenton, N. J., 3d.
HALL, Cloyes T., 1st Lieut., Morrisville, N. Y., 1st.
HART, Maurice D., 1st Lieut., St. Albans, L. I., N. Y., 2d.
HARTSTEIN, Edward, 1st Lieut., Brooklyn, 2d.
HAYMAN, Charles R., 1st Lieut., Scarsdale, N. Y., 2d.
HERMAN, Mark L., 1st Lieut., Adams, N. Y., 1st.
HOAK, Frank C., 1st Lieut., Niagara Falls, N. Y., 1st.
HOFFMAN, Sidney, 1st Lieut., Brooklyn, 2d.
HONIGMAN, Morris, 1st Lieut., Brooklyn, 2d.
HOTT, Louis R., 1st Lieut., Brooklyn, 2d.
KAHN, Bernard I., 1st Lieut., Brooklyn, 2d.
KAHN, Kenneth M., 1st Lieut., New York, 2d.
KALLEN, Arnold M., 1st Lieut., Newark, N. J., 3d.
KANENGISER, Clifford, 1st Lieut., Jersey City, N. J., 3d.
KARPEL, Bernard, 1st Lieut., New York, 2d.
KURLAND, Harry A., 1st Lieut., New York, 2d.
KWINT, Joseph A., 1st Lieut., Plainfield, N. J., 3d.
LAMB, Richard R., 1st Lieut., Farmingdale, N. Y., 1st.
LATEINER, Robert, 1st Lieut., New Rochelle, N. Y., 2d.
LABARBERA, Thomas, 1st Lieut., Brooklyn, 2d.
LEHMAN, Ernest, 1st Lieut., Brooklyn, 2d.
LEVINE, Harold N., 1st Lieut., Secaucus, N. J., 3d.
LEVY, Aaron, 1st Lieut., Brooklyn, 2d.
LEWIS, Benjamin, 1st Lieut., New York, 2d.
LEBELL, Irving, 1st Lieut., New York, 2d.
LICCIONE, William J., 1st Lieut., Syracuse, N. Y., 1st.
LIPSCHUTZ, Daniel M., 1st Lieut., New York, 2d.
LORD, William J., 1st Lieut., Albany, N. Y., 1st.
LOWE, Robert H., 1st Lieut., Bronx, N. Y., 2d.
LUCE, Henry A., 1st Lieut., Orange, N. J., 3d.
LUSTIG, Melvin, 1st Lieut., Newark, N. J., 3d.
MANFREDONIA, Charles, 1st Lieut., Brooklyn, 2d.
MARGOLIS, Hyman, 1st Lieut., Brooklyn, 2d.
MARINELLO, Carle J., 1st Lieut., Buffalo, 1st.
MARINO, Benjamin W., 1st Lieut., Brooklyn, 2d.
MARKOWITZ, Harold I., 1st Lieut., Jersey City, N. J., 3d.
McCABE, John A., 1st Lieut., Buffalo, 1st.
METCALF, Frederic U., 1st Lieut., Rockville Centre, N. Y., 2d.
MICELI, John A., 1st Lieut., Brooklyn, 2d.
MILLER, Eugene L., 1st Lieut., Newark, N. J., 3d.
MILLER, Lloyd D., 1st Lieut., Stapleton, N. Y., 1st.
MONALOY, Morris A., 1st Lieut., Clifton, N. J., 3d.
MONTELEONE, Joseph C., 1st Lieut., Brooklyn, 2d.
NAUMOFF, Joseph H., 1st Lieut., Schenectady, N. Y., 1st.
NELSON, Joseph P., 1st Lieut., Brooklyn, 2d.
PADYKULA, Stanley G., 1st Lieut., Endicott, N. Y., 1st.
PASTERNACK, Elroy, 1st Lieut., Passaic, N. J., 3d.
PRUYN, Robert M., 1st Lieut., Yonkers, N. Y., 2d.
RIZZO, Joseph J., 1st Lieut., New York, 2d.
ROBERTS, Arthur J. D., 1st Lieut., Bronx, N. Y., 2d.
ROCCO, Leo C., 1st Lieut., Red Bank, N. J., 3d.
ROSENBLUM, Meyer, 1st Lieut., Brooklyn, 2d.
SESIT, Myron F., 1st Lieut., New York, 2d.
SKURA, George, 1st Lieut., Brooklyn, 2d.
SMALL, Bernard L., 1st Lieut., Lawrence, L. I., N. Y., 2d.
SOLOMON, Saul, 1st Lieut., New York, 2d.
SPINNER, Morton H., 1st Lieut., New York, 2.
STINSON, Charles L., 1st Lieut., Buffalo, 1st.
TAYLOR, Charles W., 1st Lieut., Kings Park, N. Y., 2d.
WALLER, John V., 1st Lieut., New York, 2d.
ZEMAN, Michael S., 1st Lieut., New York, 2d.

SEVENTH CORPS AREA

Following is the initial report of names with grade, permanent address and active duty station of Medical Corps Reserve officers ordered to extended active duty by the Seventh Corps Area commander up to midnight, November 8:

DALY, John Raymond, Major, Clayton, Mo., The Executive, Third Military Area, St. Louis
SAMUEL, John Maurice, Captain, Little Rock, Ark., Recruiting Officer, U. S. Army, Little Rock, Ark.
BOWERS, Warner Fremont, Captain, Omaha, The Executive, Second Military Area, Omaha
SCHAEFERS, Richard Henry, 1st Lieut., Omaha, Commanding Officer, Fort Omaha, Neb.
RICHARDSON, Frederick Stuart, Major, Minneapolis, The Executive, First Military Area, Minneapolis.
WHITEHEAD, Robert Homer Jr., 1st Lieut., Camden, Ark., Commanding General, Fort Snelling, Minn.
HIBBERT, Russell William Jr., 1st Lieut., St. Louis, Commanding Officer, Fort Meade, S. D.
BASHAM, Charles Edward, 1st Lieut., Eureka, Kan., Commanding General, Fort Des Moines, Iowa
CODDON, Walter David, Captain, St. Paul, Commanding General, Fort Snelling, Minn.
COUGHLAN, Vernon Henry, Captain, Strawberry Point, Iowa, Commanding General, Fort Snelling, Minn.
EGBERT, Daniel Snell, Captain, Atlantic, Iowa, Commanding General, Fort Snelling, Minn.
BECKJORD, Philip Rains, 1st Lieut., Willmar, Minn., Commanding General, Fort Snelling, Minn.
BRODY, Sidney, 1st Lieut., Ottumwa, Iowa, Commanding General, Fort Snelling, Minn.
ELSWORTH, John Nelson, 1st Lieut., Bismarck, N. D., Commanding General, Fort Snelling, Minn.
FORMAN, Louis Hyman, 1st Lieut., Wichita, Kan., Commanding General, Fort Snelling, Minn.
FROGNER, Lester Sanford, 1st Lieut., Grand Marais, Minn., Commanding General, Fort Snelling, Minn.
HOLT, George Walterman, 1st Lieut., Wabasha, Minn., Commanding General, Fort Snelling, Minn.
IHLE, Charles William Jr., 1st Lieut., Cleghorn, Iowa, Commanding General, Fort Snelling, Minn.

MILLER, Samuel, 1st Lieut., Ellendale, N. D., Commanding General, Fort Snelling, Minn.
MOORE, George Ensley, 1st Lieut., Fulton, Mo., Commanding General, Fort Snelling, Minn.
MORRISON, John Robert, 1st Lieut., Carroll, Iowa, Commanding General, Fort Snelling, Minn.
STEUBE, Ronald Walter, 1st Lieut., St. Paul, Commanding General, Fort Snelling, Minn.
WITTE, Herbert Joseph, Captain, Marathon, Iowa, Commanding Officer, Fort Meade, S. D.
SMITH, Elmer Milton, 1st Lieut., State Center, Iowa, Commanding Officer, Fort Meade, S. D.
HENNING, Joseph Regnald, 1st Lieut., Ottawa Kan., Commanding Officer, Fort Leavenworth, Kan.
AVERY, Hiram Henry, Capt., Omaha, Commanding General, Fort Leavenworth, Kan.
BYERS, Walter Lewis, 1st Lieut., Sheffield, Iowa, Commanding General, Fort Snelling, Minn.
MUNGER, Horace Varnum, 1st Lieut., Lincoln, Neb., Commanding General, Fort Des Moines, Iowa
ADAMS, Marvin Edward, 1st Lieut., Clark, S. D., Commanding General, Fort Des Moines, Iowa
BURNETT, Francis Kirk, Captain, Clarinda, Iowa, Commanding General, Fort Snelling, Minn.
LIMBURG, John Irwin Jr., 1st Lieut., Jefferson, Iowa, Commanding General, Fort Snelling, Minn.
LINNER, Gunnar, 1st Lieut., Minneapolis, Commanding General, Fort Snelling, Minn.
GUNLAUGSON, Frederick Gunnar, 1st Lieut., Mankato, Minn., Commanding General, Fort Snelling, Minn.
HENDRICKSON, George Charles, Captain, Lebanon, Kan., Commanding Officer, Fort Omaha, Neb.
YAFFE, Henry Irvin, Captain, Minneapolis, Commanding General, Fort Snelling, Minn.
ROGGEN, Ivan John, 1st Lieut., Maurice, Iowa, Commanding General, Fort Snelling, Minn.
FRAZIER, Victor Eugene, Captain, Kansas City, Mo., Commanding Officer, Richards Field, Kansas City, Kan.
KADLUBOWSKI, Edmund John, 1st Lieut., LeMay, Mo., Commanding Officer, Jefferson Barracks, Mo.
PLECHAS, Nicholas Peter, 1st Lieut., Omaha, Commanding Officer, Fort Crook, Neb.
KASHA, Robert Leonard, 1st Lieut., Jefferson Barracks, Mo., Commanding Officer, Jefferson Barracks, Mo.
CLARK, Ira Delos, 1st Lieut., Casselton, N. D., Commanding General, Fort Snelling, Minn.
HANSS, Armand William, Captain, Springfield, Mo., Commanding Officer, Jefferson Barracks, Mo.
SPEIRS, Richard Everett, Captain, Dodge City, Kan., Commanding General, Fort Des Moines, Iowa
GARLINGHOUSE, Robert Orestes, 1st Lieut., Iowa City, Commanding General, Fort Snelling, Minn.
FOGLE, Robert Lee, Captain, Otterville, Mo., Commanding Officer, Jefferson Barracks, Mo.
FREEDLAND, Morris, 1st Lieut., Callender, Iowa, Commanding General, Fort Des Moines, Iowa
STEARNS, Alexander Bryce, 1st Lieut., Des Moines, Iowa, Commanding Officer, Fort Meade, S. D.
RIGGS, George Thomas, 1st Lieut., Amity, Mo., Commanding Officer, Jefferson Barracks, Mo.
KRIGSTEN, William Max, 1st Lieut., Sioux City, Iowa, Commanding General, Fort Snelling, Minn.
KLAUMAN, Benjamin Franklin, 1st Lieut., Kansas City, Mo., Commanding Officer, Fort Crook, Neb.
GRIGSBY, Kenneth Raymond, Captain, Medicine Lodge, Kan., Commanding Officer, Fort Crook, Neb.
JAMES, Louis Daniel, 1st Lieut., Oxford, Neb., Commanding General, Fort Benning, Ga.
DARNELL, Thomas Ficklin, 1st Lieut., Macon, Mo., Commanding General, Fort Snelling, Minn.
MATASSARIN, Frederick William, 1st Lieut., Wichita, Kan., Commanding General, Fort Benning, Ga.
COOPER, Elven Ross, Captain, Warrensburg, Mo., Commanding Officer, Jefferson Barracks, Mo.
HAUKENBERRY, Everett Francis, Captain, Stockdale, Kan., Commanding General, Fort Benning, Ga.
HUGHES, Max, 1st Lieut., Walnut Ridge, Ark., Commanding General, Fort Benning, Ga.
WOOD, Turner Anderson, 1st Lieut., Conway, Ark., Commanding General, Fort Benning, Ga.
THOMAS, Horace Edward, 1st Lieut., Columbia, Mo., Commanding General, Fort Benning, Ga.
ROBERTS, Howard Emerson, 1st Lieut., Topeka, Kan., Commanding Officer, Camp Joseph T. Robinson, Ark.
VANDOVER, John Taylor, 1st Lieut., St. Louis, Commanding General, Fort Benning, Ga.
KNOLL, William, 1st Lieut., McLaughlin, S. D., Commanding General, Fort Benning, Ga.
RAY, Richard Horace, 1st Lieut., Willow Springs, Mo., Commanding General, Fort Benning, Ga.
CARLSON, Marlin Winthrop, 1st Lieut., Ellinwood, Kan., Commanding General, Fort Benning, Ga.
BUHL, Clayton Edwin, 1st Lieut., Mullen, Neb., Commanding Officer, Jefferson Barracks, Mo.
SMITH, James Turner, 1st Lieut., Paris, Ark., Commanding General, Fort Benning, Ga.
HUSKINS, James Daniel, 1st Lieut., Siloam Springs, Ark., Commanding General, Fort Benning, Ga.
KELLY, Miles Fountain, 1st Lieut., Sheridan, Ark., Commanding Officer, Jefferson Barracks, Mo.
VAUBEL, Ellis Kendall, 1st Lieut., Peterson, Iowa, Commanding General, Fort Benning, Ga.
HELLWEG, Charles Edward, Captain, Mount Vernon, Mo., Commanding Officer, Jefferson Barracks, Mo.
SLAUGHTER, Wilbur Logan, Captain, Clarksville, Ark., Commanding General, Fort Benning, Ga.
BANET, Samuel Richard, 1st Lieut., St. Louis, Commanding General, Fort Benning, Ga.
FRY, Gerald Alba, 1st Lieut., Wichita, Kan., Commanding General, Fort Benning, Ga.
GREENBAUM, Roy, 1st Lieut., St. Louis, Commanding General, Fort Benning, Ga.
JACKSON, Jabez Fenton, 1st Lieut., Walnut Ridge, Ark., Commanding General, Fort Benning, Ga.
SHADE, Virgil Edward, Captain, Liberty, Mo., Commanding General, Fort Benning, Ga.
ZINSCHLAG, Edward Niehoff, 1st Lieut., Glendale, Mo., Commanding General, Fort Benning, Ga.
THOMPSON, Elvin Dean, 1st Lieut., Webster City, Iowa, Commanding General, Fort Benning, Ga.
WALLACE, Edwin Sharp, Captain, Lexington, Mo., Commanding General, Fort Benning, Ga.
McGEE, William Joseph, 1st Lieut., Eldorado, Mo., Commanding General, Fort Benning, Ga.
DILLON, John Alfred Jr., Captain, Great Bend, Kan., Commanding General, Fort Benning, Ga.
GRIMES, Burton Piper, 1st Lieut., St. Peter, Minn., Commanding General, Fort Benning, Ga.
BINNS, Byron Zack, 1st Lieut., Monticello, Ark., Commanding General, Fort Benning, Ga.
KLIPPEN, Arthur J., 1st Lieut., St. Louis, Commanding General, Fort Benning, Ga.
WEINBERG, Harry B., 1st Lieut., Davenport, Iowa, Commanding General, Fort Benning, Ga.
BURBRIDGE, Glen E., 1st Lieut., Logan, Iowa, Commanding General, Fort Benning, Ga.

NAVAL RESERVE OFFICERS ORDERED TO ACTIVE DUTY

Following is a list of naval reserve officers who have been called to active duty, together with a record of their grade, permanent address and station to which they have been sent. THE JOURNAL proposes to print regularly the announcements concerning reserve officers assigned to active duty both in the Army and in the Navy Medical Corps.

ABRAHAMSON, Robert H., Lieut. M. C.-V. (S.), New York, Naval Recruiting Station, Albany, N. Y.
ADAMS, Thomas S., Lieut. Comdr. M. C.-O., Jacksonville, Fla., Naval Reserve Armory, Jacksonville, Fla.
AGNEW, John R., Lieut. M. C.-V. (S.), Springfield, Mass., Marine Recruiting Station, Springfield, Mass.
ALBRITTAIN, J. W., Lieut. (j. g.) M. C.-O., Baltimore, Norfolk Naval Hospital
ALDRICH, Leonard C., Lieut. (j. g.) M. C.-O., Houghton, Mich., Hancock, Mich.
ARNOLD, H. B., Lieut. Comdr. M. C.-O., New Haven, Conn., Naval Reserve Armory, New Haven, Conn.
BAILEY, Byron G., Lieut. M. C.-V. (S.), San Diego, Calif., M. C. B., San Diego, Calif.
BAKST, Henry J., Lieut. (j. g.) M. C.-V. (S.), Brookline, Mass., Naval Hospital, Boston.
BARNER, Henry Adolph, Lieut. Comdr. M. C.-V. (G.), Bremerton, Wash., N. Yd., Puget Sound, Wash.
BARNSHAW, Harold D., Lieut. M. C.-O., Merchantville, N. Y., N. H., Pensacola, Fla.
BARON, Abraham Isaac, Lieut. Comdr. M. C.-V. (S.), Jasper, Texas, N. H., Chelsea, Mass.
BARTON, Edward William Jr., Lieut. (j. g.) M. C.-V. (S.), San Gabriel, Calif., 11th Naval District
BASSETT, Robert B., Lieut. (j. g.) M. C.-O., St. Louis, N. R. A. B., St. Louis (Robertson)
BELL, Fredric W., Lieut. M. C.-V. (S.), Warrington, Fla., N. H., Pensacola, Fla.
BERTRAM, Albert J., Lieut. Comdr. M. C.-V. (S.), Miami, Fla., Naval Reserve Armory, Miami, Fla.
BIBLER, Lester D., Lieut. M. C.-O., Indianapolis, 16th Bn., U. S. M. C. R., Indianapolis
BIGELOW, Robert B., Lieut. Comdr. M. C.-V. (S.), Boston, N. A. S., Pensacola, Fla.
BIRD, James D. Jr., Lieut. M. C.-V. (S.), Wheeling, W. Va., N. H., San Diego, Calif.
BLEASBY, Charles B., Lieut. Comdr. M. C.-O., Garfield, N. J., N. T. S., N. O. B., Norfolk, Va.
BOGGS, Robert, Lieut. M. C.-V. (G.), New York, U. S. S. Illinois

BOSTIC, Sam Crawford, Lieut. M. C.-V. (G.), Flushing, N. Y., 3d Naval District

BOWEN, Harold J., Lieut. Comdr. M. C.-O., Charleston, S. C., Naval Reserve Armory, Charleston, S. C.

BOWERS, John Z., Lieut. (j. g.) M. C.-O., Baltimore, Norfolk Naval Hospital

BOWSER, Frank E., Lieut. M. C.-V. (G.), Key West, Fla., N. Sta., Key West, Fla.

BRADY, Alfred S. Jr., Lieut. M. C.-V. (S.), Charleston, W. Va., N. T. S., Newport, R. I.

BREUCHER, William, Lieut. Comdr. M. C.-O., Philadelphia, 6th Bn., U. S. M. C. R., Philadelphia

BROOKS, Morris, Lieut. Comdr. M. C.-V. (S.), Brooklyn, Navy Yard, New York

BROWN, Byron F., Lieut. Comdr. M. C.-O., Hopedale, Mass., N. H., Newport, R. I.

BROWN, John Joseph, Lieut M. C.-V. (S.), Bellerose, N. Y., Rec. Ship (U. S. S. Camden), Brooklyn

BUCKLEY, Clarence J., Lieut. Comdr. M. C.-V. (S.), Cleveland, Navy Yard, Philadelphia

BULLWINKEL, Harry G., Lieut. Comdr. M. C.-O., Garden City, N. Y., N. H., Brooklyn

BURRIS, Floyd L., Lieut. (j. g.) M. C.-O., Michigan City, Ind., Naval Reserve Armory, Michigan City, Ind.

BYERS, Philip Larkin, Lieut. (j. g.) M. C.-V. (G.), Kansas City, Mo., N. R. S., Kansas City, Mo.

CARDWELL, John L., Lieut. Comdr. M. C.-O., Washington, D. C., N. H., Washington, D. C.

CARMODY, Robert F., Lieut. M. C.-O., Brookline, Mass., 2d Bn., U. S. M. C. R., Boston

CARSON, James G., Lieut. (j. g.) M. C.-V. (G.), San Francisco, 12th Naval District

CHAPMAN, Sims A., Lieut. Comdr. M. C.-O., New Orleans, N. H., Pensacola, Fla.

CHURCHILL, Asa G., Lieut. Comdr. M. C.-O., Coronado, Calif., N. H., San Diego, Calif.

CLARK, Irving T., Lieut. (j. g.) M. C.-V. (G.), Opa Locka, Fla., N. A. S, Miami, Fla.

CLIFFORD, Richard V., Lieut. (j. g.) M. C.-V. (G.), Drexel Hill, Pa., N. H., Philadelphia

COLLINS, R. Bruce, Lieut. (j. g.) M. C.-V. (G.), Rock Island, Ill., N. A. S., Pensacola, Fla.

CONNELL, DeBert, W., Lieut. (j. g.) M. C.-O., Berkeley, Calif., N. H., Mare Island, Calif.

COOK, Enos P., Lieut. Comdr. M. C.-O., San Jose, Calif., N. H., Mare Island, Calif.

COOPER, Howard N., Lieut. M. C.-O., Watertown, N. Y., N. T. S., Newport, R. I.

COUNTS, William R., Lieut. (j. g.) M. C.-V. (G.), Amonate, Va., N. R. A. B., Anacostia, D. C.

COX, Russell M., Lieut. Comdr. M. C.-O., Portsmouth, Va., Norfolk Naval Hospital

CRANDALL, Bradford B., Lieut. (j. g.) M. C.-M., Mansfield, Conn., Navy Yard Disp., Philadelphia

CRUICKSHANK, Frank S., Lieut. M. C.-V. (S.), Brookline, Mass., Navy Yard Disp., Boston

CUPAIUOLI, Richard A., Lieut. (j. g.) M. C.-V. (G.), North Bergen, N. J., 4th Bn., U. S. M. C. R., Newark, N. J.

CUSHING, Edward H., Lieut. Comdr. M. C.-V. (S.), Bratenahl, Ohio, Bu. M. & S., Navy Dept., Washington, D. C.

DALY, Sylvester, Lieut. M. C.-V. (S.), Westwood, N. J., N. R. A. B., Floyd Bennett Field, N. Y.

DANIELS, Donald Howard, Lieut. M. C.-O., Portland, Me., Naval Reserve Armory, Portland, Me.

DANNA, Pascal L., Lieut. M. C.-V. (S.), New Orleans, N. R. S., New Orleans

DAVIES, Drew L., Lieut. M. C.-O., Columbus, Ohio, Columbus, Ohio

DAVIS, William J. N., Lieut. Comdr. M. C.-V. (G.), Chicago, N. R. Signal School, Chicago

DE CICCO, Luigi M., Lieut. Comdr. M. C.-V. (S.), Fitchburg, Mass., Navy Yard, Portsmouth, N. H.

DE COSTA, Edwin J., Lieut. M. C.-O., Chicago, 9th Bn., U. S. M. C. R., Chicago

DEMENT, Donald E., Lieut. Comdr. M. C.-V. (G.), Squantum, Mass., N. R. A. B., Boston

de YOANNA, Saverio A., Lieut. Comdr. M. C.-V. (S.), Brooklyn, Navy Yard Disp., New York

DICKINSON, Everett H., Lieut. Comdr. M. C.-V. (G.), Ardmore, Pa., N. H. Philadelphia

DOBBS, Frederick H., Lieut (j. g) M. C.-V. (G.), Wheeling, W. Va., N. A. S., Pensacola, Fla.

DOBBS, Otto Ross, Lieut. Comdr. M. C.-V. (S.), St. Louis, N. R. S., St. Louis

DOBSON, John P., Lieut. (j. g.) M. C.-V. (G.), Manhasset, N. Y., N. A. S., Pensacola, Fla.

DOCKRY, Lyman Edwin, Comdr. M. C.-V. (G.), Great Lakes, Ill., N. T. S., Great Lakes, Ill.

DOYLE, Joseph Bernard, Lieut. (j. g.) M. C.-V. (S.), Newton, Mass., N. H., Chelsea, Mass.

DRAVO, Emory Low, Lieut. Comdr. M. C.-V. (S.), Great Lakes, Ill., N. T. S., Great Lakes, Ill.

ELLETT, William H., Lieut. M. C.-O., Appleton City, Wis., N. H., San Diego, Calif.

ENGLISH, Glenn G., Lieut. M. C.-O., Los Angeles, 13th Bn., U. S. M. C. R., Los Angeles

FAKEHANY, Joseph P., Lieut. (j. g.) M. C.-V. (G.), Toledo, Ohio, Toledo, Ohio

FERGUSON, R. S., Lieut. Comdr. M. C.-V. (S.), New York, Norfolk Naval Hospital

FLYNN, John R., Lieut. (j. g.) M. C.-V. (G.), Tacoma, Wash., 13th Naval District, Seattle

FORBES, Alexander, Lieut. Comdr. M. C.-V. (S.), Milton, Mass., N. A. S., Pensacola, Fla.

FORD, Richard B., Lieut. M. C.-V. (S), Tulsa, Okla., N. H., Pensacola, Fla.

FOX, Frederick G., Lieut. Comdr. M. C.-V. (S.), Chicago, M. B., Quantico, Va.

FOX, M. R., Lieut. Comdr. M. C.-V. (S.), Portsmouth, Va., Norfolk Naval Hospital

FREYMANN, John J., Lieut. Comdr. M. C.-V. (G.), Omaha, N. R. S., Omaha

FRONEK, Otis John, Lieut. (j. g.) M. C.-V. (G.), University Heights, Ohio, N. R. S., Cleveland

FUQUA, Samuel A., Lieut. Comdr. M. C.-V. (G.), Chicago, N. T. S., Great Lakes, Ill.

GARNETT, Herman M., Lieut. M. C.-V. (G.), Long Island City, N. Y., Rec. Ship, New York

GATES, Donald C., Lieut.. M. C.-V. (S.), Boston, N. A. S., Pensacola, Fla.

GAYNOR, Edmund P., Lieut. (j. g.) M. C.-O., San Francisco, N. H., Mare Island, Calif.

GERNARD, Henry C., Lieut. M. C.-O., Los Angeles, Naval Dispensary, Long Beach, Calif.

GERSTLE, Mark L. Jr., Lieut. M. C.-V. (S.), Cupertino, Calif., 11th Naval District, San Diego, Calif.

GILBERT, Allen C., Lieut. Comdr. M. C.-V. (S.), Dallas, Texas, Norfolk Navy Yard

GINDHART, Floyd D., Lieut. M. C.-O., New York, N. H., Brooklyn

GOLDBLATT, Harold W., Lieut. (j. g.) M. C.-V. (G.), Lawrence, Mass., Navy Yard Disp., Boston

GOLDSMITH, Harry, Lieut. Comdr. M. C.-V. (S.), Baltimore, M. B., Quantico, Va.

GOLLER, John J., Lieut. Comdr. M. C.-O., Staten Island, N. Y., N. H., Newport, R. I.

GRAYBIEL, Ashton, Lieut. Comdr. M. C.-V. (S.), Brookline, Mass., N. A. S., Pensacola, Fla.

GROW, W. L., Lieut. Comdr. M. C.-V. (S.), Bonita, Calif., U. S. S. Rigel

GRUGGEL, John S., Lieut. M. C.-M., New York, Sub. Base, New London, Conn.

HAGUE, Robert F., Lieut. M. C.-V. (G.), Flint, Mich., N. T. S., Great Lakes, Ill.

HALL, Frank McK., Lieut. M. C.-V. (G.), West Lafayette, Ind., U. S. S. Sacramento

HALPERIN, Meyer A., Lieut. Comdr. M. C.-V. (S.), Brooklyn, N. H., Brooklyn

HAMER, Jerome B., Lieut. (j. g.) M. C.-V. (G.), Charlotte, N. C., Marine Corps Res., Charlotte, N. C., 21st Bn.

HAMMERSLEY, Eugene R., Lieut. M. C.-V. (G.), Tuscarawas, Ohio, F. M. F., M. B., Quantico, Va.

HAMMOND, Jack, Lieut. (j. g.) M. C.-O., New York, N. H., Chelsea, Mass.

HAMMOND, T. V. Jr., Lieut. Comdr. M. C.-V. (G.), Meadows, Md., Navy Yard, Portsmouth, N. H.

HANTEN, John Sylvester, Lieut. M. C.-V. (S.), San Francisco, N. H., Mare Island, Calif.

HARBACH, Francis O., Lieut. M. C.-V. (S.), Fayetteville, N. Y., M. R. S., Syracuse, N. Y.

HARKINS, Herbert P., Lieut. (j. g.) M. C.-V. (G.), Melrose Park, Pa., N. H., Philadelphia

HARRIS, Sydney L., Lieut. M. C.-O., Pelham Manor, N. Y., Nav. Res. Radio School, Noroton, Conn.

HAUMAN, Hazen L., Lieut. (j. g.) M. C.-O., Toledo, Ohio, 8th Bn., U. S. M. C. R., Toledo, Ohio

HAWES, John K., Lieut. M. C.-V. (G.), Brooklyn, N. R. A. B., New York (Brooklyn)

HELGESSON, Uno, H., Lieut. Comdr., M. C.-V. (S.), Northampton, Mass., N. H., Washington, D. C.

HERNDON, Ray F., Lieut. Comdr. M. C.-O. Retired, Houston, Texas, N. R. S., Dallas, Texas

HITCH, Joseph Martin, Lieut. (j. g.) M. C.-V. (S.), Raleigh, N. C., N. R. S., Raleigh, N. C.

HOCKETT, Verden Edw., Lieut. M. C.-V. (G.), Salem, Ore., State Med. Off. with Director of Selective Serv. State of Oregon

HODGES, Francis T., Lieut. (j. g.) M. C.-V. (G.), San Francisco, 12th Naval District

HOFER, Clarence J. M., Lieut. M. C.-O., Matuchen, N. J., N. T. S., Newport, R. I.

HOGEBOOM, Willard L., Lieut. (j. g.) M. C.-O., Helmuth, N. Y., Norfolk Naval Hospital

HOLM, Alf J., Lieut. Comdr. M. C.-V. (S.), Chicago, N. T. S., Great Lakes, Ill.

HOMAN, Ralph H., Lieut. Comdr. M. C.-V. (S.), El Paso, Texas, N. H., Parris Island, S. C.

HUMMEL, Merwin L., Lieut. M. C.-O., Merchantville, N. J., N. R. A. B., Cape May, N. J.

HUNGATE, Carroll P., Lieut. M. C.-V. (G.), Kansas City, Mo., 8th Bn., U. S. M. C. R.

ICKS, Karl, Lieut. M. C.-O., Green Bay, Wis., Green Bay, Wis.

IVES, Warren C., Lieut. Comdr. M. C.-V. (G.), Rockford, Ill., Sub. Base, New London, Conn.

JABLONS, Abraham, Lieut. Comdr. M. C.-V. (G.), New York, N. A. S., Norfolk, Va.

JACKSON, Harry H., Lieut. Comdr. M. C.-V. (G.), Detroit, N. R. A. B., Grosse Isle, Mich.

JACKSON, Waddie P., Comdr. M. C.-V. (G.), Roanoke, Va., N. A. S., Norfolk, Va.

JACOBSON, Sheldon A., Lieut. M. C.-V. (G.), Brooklyn, N. H., Philadelphia

JAMES, Gordon M., Lieut. M. C.-V. (G.), Bremerton, Wash., Navy Yard Disp., Bremerton, Wash.
JEWETT, Everett Porter, Lieut. Comdr. M. C.-O., Worcester, Mass., Naval Reserve Armory, Worcester, Mass.
JOHNSRUD, Russell L., Lieut. (j. g.) M. C.-O., Portland, Ore., N. R. S., Portland, Ore.
JOSEPHS, Louis, Lieut. Comdr. M. C.-V. (S.), Los Angeles, M. C. B., San Diego, Calif.
JUNKIN, William McC., Lieut. Comdr. M. C.-V. (S.), Fredericksburg, Va., N. T. S., Norfolk, Va.
KAHN, Gustav M., Lieut. (j. g.) M. C.-O., Galveston, Texas, 15th Bn., U. S. M. C. R., Galveston, Texas
KECK, Everett B., Lieut. Comdr. M. C.-V. (S.), Madison, Wis., M. B., Quantico, Va.
KECK, Herman, Lieut. M. C.-O., Cincinnati, N. H., San Diego, Calif.
KENNEY, LeRoy, L., Lieut. Comdr. M. C.-V. (S.), Kerrville, Texas, N. H., Pensacola, Fla.
KLEIN, Leonard, Lieut. (j. g.) M. C.-V. (G.), New York, N. R. S., New York
KNOWLTON, Don S., Lieut. Comdr. M. C.-V. (S.), Washington, D. C., 5th Bn., U. S. M. C. R., Washington, D. C.
KORNBLUT, Alfred, Lieut. M. C.-O., Bridgeport, Conn., N. H., Pensacola, Fla.
KREPELA, Miles C., Lieut. (j. g.) M. C.-V. (S.), New York, U. S. S. Illinois
LARSON, Lawrence M., Lieut. M. C.-O., Minneapolis, N. R. A. B., Minneapolis
LAUTERBACH, Chester H., Lieut. (j. g.) M. C.-O., Rochester, N. Y., N. H., Chelsea, Mass.
LEBENSOHN, James E., Lieut. Comdr. M. C.-V. (G.), Chicago, N. R. S., Chicago
LEETE, Edward D., Lieut. Comdr. M. C.-V. (G.), Boston, N. R. S., Boston
LELAND, John A. C., Lieut. (j. g.) M. C.-V. (G.), Minneapolis, N. H., Mare Island, Calif.
LELAND, Sherman, Lieut. M. C.-O., San Francisco, N. H., Mare Island, Calif.
LEWIS, Will E., Lieut. (j. g.) M. C.-O., Tacoma, Wash., Naval Reserve Recruiting Office, Tacoma, Wash.
LIEBER, Hyman, Lieut. M. C.-V. (S.), Flushing, N. Y., Navy Yard, New York
LILJENCRANTZ, Eric, Comdr. M. C.-V. (S.), San Francisco, Bu. Aero., Navy Dept., Washington, D. C.
LIPPSCHUTZ, Eugene J., Lieut. (j. g.) M. C.-O., Williamsville, N. Y., Norfolk Naval Hospital
LONGENECKER, Charles R., Lieut. (j. g.) M. C.-O., New York, N. T. S., Newport, R. I.
LORENZ, George Jr., Lieut. Comdr. M. C.-V. (S.), Glenside, Pa., N. H., Philadelphia
LUFKIN, N. H., Lieut. M. C.-V. (S.), Minneapolis, 18th Bn., St. Paul
MacGOWAN, Birkhead, Lieut. Comdr. M. C.-V. (S.), Baltimore, Naval Reserve Armory, Baltimore, and 1st Bn., Naval Reserve
MACNISH, James M., Lieut. (j. g.) M. C.-O., Webster Groves, Mo., N. H., San Diego, Calif.
MARSDEN, George A., Lieut. Comdr. M. C.-O., Oswego, N. Y., N. T. S., Newport, R. I.
MARTIN, William L, Comdr. M. C.-O., Merion Station, Pa., N. H., Philadelphia
MASON, Malcolm W., Lieut. M. C.-V. (S.), Elmhurst, N. Y., Third Naval District
MAYNE, Roy Malone, Lieut. Comdr. M. C.-O., Duluth, Minn., U. S. S. Paducah
McCARTHY, Donald, Lieut. Comdr. M. C.-V. (G.), Minneapolis, N. R. S., Minneapolis
McCRIMMON, Herman P., Lieut. Comdr. M. C.-V. (S.), Great Lakes, Ill., N. T. S., Great Lakes, Ill
McCULLOUGH, Clifford P., Lieut. Comdr. M. C.-V. (S.), Lake Forest, Ill., N. T. S, Great Lakes, Ill.
McKEEVER, Duncan C., Lieut. M. C.-O., Houston, Texas, Naval Reserve Armory, Houston, Texas.
McKITTERICK, John C., Lieut. Comdr. M. C.-O., Burlington, Iowa, Burlington, Iowa
McLEAN, Marvin Mc.D, Lieut. Comdr. M. C.-V. (S.), Washington, D. C., Navy Yard, Washington, D. C.
McMAHON, Henry E., Lieut. M. C.-V. (G.), Plandome, N. Y., N. H., Brooklyn
McNAMARA, D. H., Lieut. (j. g.) M. C.-O., Santa Barbara, Calif., Naval Disp., Long Beach, Calif.
MEADOR, James R., Lieut. (j. g.) M. C.-O., Clayton, Mo., N. H., San Diego, Calif.
MECK, Floyd S., Lieut. M. C.-V. (G.), Cleveland Heights, Ohio, N. R. S., Cleveland
MERRITT, Edward L., Lieut. Comdr. M. C.-V. (S.), Fall River, Mass., N. H., Newport, R. I.
MEYERS, Frederick C., Lieut. M. C.-O., Briarcliff Manor, N. Y., Norfolk Naval Hospital
MILLER, Hugh A, Jr., Lieut. (j. g.) M. C.-O., Detroit, U. S. S. Dubuque
MILLER, LaFayette T., Lieut. Comdr. M. C.-O., Dallas, Texas, Naval Reserve Armory, Dallas, Texas
MINTZ, Emanuel R., Lieut. M. C.-V. (S.), Boston, N. H., Chelsea, Mass.
MITCHELL, John T., Lieut. (j. g.) M. C.-O., Miami, Fla., N. H., Pensacola, Fla.
MITCHELL, Marvin A., Lieut. (j. g.) M. C.-O., Atlanta, Ga., Naval Reserve Armory, Atlanta, Ga.
MITCHELL, William G., Lieut. Comdr. M. C.-V. (S.), San Angelo, Texas, N. H., Parris Island, S. C.
MOLESKI, Stanley L., Lieut. (j. g.) M. C.-V. (G.), Grand Rapids, Mich., N. H., Great Lakes, Ill
MOORE, Moore, Jr., Lieut. Comdr. M. C.-V. (G.), Beckley, W. Va., N. H., Charleston, S. C.
MOSELEY, Lonnie B., Lieut. M. C.-V. (S.), Jackson, Miss., N. H., Parris Island, S. C.
MULLEN, Edward A., Lieut. Comdr. M. C.-O., Philadelphia, N. H., Pensacola, Fla.
MURRAY, Ray W., Lieut. Comdr. M. C.-V. (S.), Washington, D. C., N. H., Washington, D. C.
NELSON, Edwin R., Lieut. (j. g.) M. C.-V. (G.), Madison, Wis., N. H., Bremerton, Wash.
NICHOLS, Samuel I., Lieut. (j. g.) M. C.-V. (S.), Norfolk, Va., (Instr. N. A. S., Pensacola)
NORCROSS, Nathan C., Lieut. M. C.-V. (S.), Cambridge, Mass., N. H., Philadelphia
NORTHINGTON, Page O., Comdr. M. C.-O., Garden City, N. Y., N. H., Brooklyn
NORTHUP, Spencer W., Lieut. (j. g.) M. C.-O., Toledo, Ohio, N. T. S., San Diego, Calif.
NUCKOLLS, John B., Lieut. M. C.-V. (S.), Charleston, S. C., N. H., Charleston, S. C.
ORMSBY, Edward B., Lieut. Comdr. M. C.-V. (S.), Boston, N. H., Chelsea, Mass.
O'TOOLE, John B., Jr., Lieut. (j. g.) M. C.-V. (G.), New Bedford, Mass., N. T. S., Newport, R. I.
PAGAN, Albert E., Lieut. Comdr. M. C.-V. (S.), Washington, D. C., Navy Yard, Washington, D. C.
PARKER, Arthur D., Lieut. Comdr. M. C.-V. (S.), Norfolk, Va., N. T. S., N. O. B., Norfolk, Va.
PARKER, Thomas, Lt. (j. g.) M. C.-V. (S.), Greenville, S. C., Navy Yard, Charleston, S. C.
PARSONS, William C., Lieut. M. C.-O., Birmingham, Ala., Naval Reserve Armory, Birmingham, Ala.
PEEK, John S., Lieut. (j. g.) M. C.-V. (G.), Brownsville, Texas., N. A. S., Pensacola, Fla.
PENTZ, Clarence R., Lieut. M. C.-O., Long Beach, Calif., N. R. A. B., Long Beach, Calif.
PEPPER, Milton, Lieut. (j. g.) M. C.-V. (S.), Salt Lake City, N. R. S., Salt Lake City
PETERS, John R., Lieut. Comdr. M. C.-V. (S.), Pewee Valley, Ky., N. T. S., Great Lakes, Ill.
PETRIK, Albert C., Lieut. Comdr. M. C.-V. (S.), Tuscaloosa, Ala., Naval Stat., Key West, Fla.
PETRONE, Francis J., Lieut. M. C.-V. (G.), Fall River, Mass., N. A. S., Quoinset Point, R. I.
PHILBROOK, Frank R., Lieut. M. C.-V. (S.), Somerville, Mass., N. R. A. B., Squantum, Boston
PIEPER, Howard C., Lieut. (j. g.), M. C.-V. (G.), Long Branch, N. J., N. R. A. B., Chicago (Glenview), Ill.
POTTER, Leo E., Lieut. M. C.-V. (S.), Houston, Texas, N. R. S., Houston, Texas
PRICE, Richard H., Lieut. Comdr. M. C.-V. (S.), Quantico, Va., N. H., Parris Island, S. C.
PUTTLER, Simpson D., Lieut. (j. g.) M. C.-V. (G.), Grosse Ile, Mich., N. R. A. B., Grosse Ile., Detroit
RAVEL, Vincent M., Lieut. (j. g.) M. C.-V. (G.) El Paso, Texas, N. R. S., Dallas, Texas
REDDING, John O., Lieut. M. C.-O., New Orleans, 10th Bn., U. S. M. C. R., New Orleans
REGAN, James J., Lieut. Comdr. M. C.-V. (G.), Boston, N. H., Chelsea, Mass.
RICHARD, Harold J., Lieut. Comdr. M. C.-V. (S.), Pensacola, Fla., N. A. S., Pensacola, Fla.
ROBERTON, Charles G., Lieut. Comdr. M. C.-V. (G.), San Diego, Calif., M. C. B., San Diego, Calif.
ROBERTS, Melvin D., Lieut. Comdr. M. C. Retired, Hancock, Mich., M. R. S., Salt Lake City.
ROBERTSON, Robert F., Lieut. (j. g.) M. C.-V. (G.), Los Angeles, N. H., San Diego, Calif.
ROBINSON, Nathaniel D., Lieut. (j. g.) M. C.-V. (G.), Pawtucket, R. I., N. R. A. B., Kansas City, Kan.
ROONEY, James S., Lieut. Comdr. M. C.-V. (S.), Newton, Mass., N. H., Chelsea, Mass.
ROSEN, Abner, Lieut. (j. g.) M. C.-V. (G.), New York, Nav. Torp. Sta., Newport, R. I.
ROSEN, Max, Lieut. M. C.-V. (S.), Brooklyn Navy Yard, New York.
ROSENBLOOM, Monroe A., Lieut. M. C.-V. (S.), New York, Third Naval District
ROSSER, Marion T., Lieut. Comdr. M. C.-V. (S.), Parris Island, S. C., Norfolk Naval Hospital.
ROWOHLT, George O., Lieut. (j. g.) M. C.-O., Dumont, N. J., Norfolk Naval Hospital
SALA, Roland O., Lieut. Comdr. M. C.-V. (G.) Rock Island, Ill., N. A. S., Pensacola, Fla.
SALING, Robert P., Lieut. (j. g.) M. C.-V. (G.), Pittsburgh, N. H., Philadelphia
SASKA, August, Lieut. Comdr. M. C.-O., Pittsburgh, M. R. S., Pittsburgh
SASSMAN, John J., Lieut. (j. g.) M. C.-V. (G.), Allentown, Pa., N. A. S., San Diego, Calif.
SAVERIEN, Arnold E., Lieut. Comdr. M. C.-O., Los Angeles, Naval Disp., Long Beach, Calif.
SAXL, Newton T., Lieut. Comdr. M. C.-V. (S.), New York, N. H., Brooklyn
SCHACHTER, H. A. H., Lieut. Comdr. M. C.-V. (S.), Newark, N. J., N. H., Brooklyn
SHAFER, William L. Jr., Lieut. Comdr. M. C.-O., Alexandria, Va., 5th Bn., U. S. M. C. R., Washington, D. C.
SCOTT, Edgar M., Jr., Lieut. M. C.-V. (S.), Birmingham, Ala., N. R. S., Birmingham, Ala.
SEGAL, Samuel, Jr., Lieut. Comdr. M. C.-O., Springfield, Mass., Naval Reserve Armory, Springfield, Mass.
SHELDON, Suel A., Lieut. Comdr. M. C.-O., Saginaw, Mich., Saginaw, Mich.
SHER, Joseph J., Lieut. M. C.-V. (S.), Brooklyn, N. H., Brooklyn
SHONE, Lloyd B., Lieut. Comdr. M. C.-V. (G.), San Francisco, Headquarters, 12th Naval District

SIAU, James R. S., Jr., Lieut. (j. g.) M. C.-O., Georgetown, S. C., Naval Reserve Armory, Georgetown, S. C.
SIMPSON, William A., Lieut. Comdr. M. C.-V. (S.), Baltimore, Navy Yard, Norfolk, Va.
SISSON, Merrell A., Lieut. (j. g.) M. C.-O., Portland, Ore., 20th Bn., U. S. M. C. R, Portland, Ore
SMART, William R., Lieut. (j. g.) M. C.-V. (G.), Oklahoma City, N. H., Parris Island, S. C.
SMITH, Arthur B. R., Lieut. M. C.-V. (S.) Jersey City, N. J., Sub. Base, New London, Conn.
SOURS, James W., Lieut. (j. g.) M. C.-O., Peoria, Ill., N. T. S., San Diego, Calif.
SOUTHCOMBE, Robert H., Lieut. M. C.-O., Spokane, Wash., 14th Bn., U. S. M. C. R., Spokane, Wash.
SOUTHWORTH, V. C., Lieut. Comdr. M. C.-V. (S), Cambridge, Md., N. H., Pensacola, Fla.
SPEER, Frederic A., Lieut. (j. g.) M. C.-O., Kansas City, Kan., N. H., San Diego, Calif.
SPEER, Leland N., Lieut. (j. g.) M. C.-O., Kansas City, Mo., N. H., San Diego, Calif.
STEDMAN, Harold E., Lieut. M. C.-V. (G.), Hempstead, N. Y., N. H., Brooklyn
STEIN, Emil J., Comdr., M. C.-A., Chicago, N. T. S., Great Lakes, Ill.
STEINBERG, Miguel, Lieut. Comdr. M. C.-V. (S.), New York, N. H., Brooklyn
STEWART, Wayne H., Lieut. (j. g.) M. C.-O., Coraopolis, Pa., N. T. S., N. O. B., Norfolk, Va.
STOOPS, William A., Comdr. M. C.-V. (G.), Newport, R. I., N. H., Newport, R. I.
STUTTLE, Fred L., Lieut. (j. g.) M. C.-V. (G.), Peoria, Ill., Peoria, Ill.
THOMAS, George C., Lieut. Comdr. M. C.-V. (G.), Mifflinburg, Pa., Navy Yard, Philadelphia
THOMAS, Wesley C., Lieut. Comdr. M. C.-V. (S.), Charleston, W. Va., N. H., Parris Island, S. C.
THURSTON, H. S., Lieut. Comdr. M. C.-O. Retired, Indianapolis, Naval Reserve Radio School, Indianapolis
TIPTON, Van C., Lieut. M. C.-V. (S.), Austin, Texas, N. R. S., Macon, Ga.
TORLAND, Paal, Lieut. Comdr. M. C.-V. (S.), San Diego, Calif., N. T. S., San Diego, Calif.
TROENSEGAARD, Clement C., Lieut. Comdr. M. C.-V. (S.), Pasadena, Calif., N. R. A. B., Oakland, Calif.
UNDERWOOD, George R., Lieut. (j. g.) M. C.-V. (S.), Lincoln, Neb., Naval Reserve Armory, Omaha
VIEIRA, Edwin, Lieut. (j. g.) M. C.-V. (G.) East Providence, R. I., Nav. Torp. Sta., Newport, R. I.
WALD, Oscar, Lieut. Comdr. M. C.-V. (S.), Brooklyn, N. R. S., New York
WARD, Robert H., Lieut. (j. g.) M. C.-V. (G.), Boston, N. A. S., Pensacola, Fla.
WARM, Herbert, Lieut. (j. g.) M. C.-V. (G.), Hamilton, Ohio, N. R. S., Cincinnati
WARREN, Jack, Lieut. (j. g.) M. C.-V. (G.), Tahatchi, N. M., Yd., Disp., Puget Sound, NYd., Bremerton, Wash.
WARREN, Richard, Lieut. (j. g.) M. C.-O., Boston, N. H., Chelsea, Mass.
WASSELL, Corydon M., Lieut. Comdr. M. C.-V. (S.), Key West, Fla., Naval Station, Key West, Fla.
WATKINS, Harry C., Jr., Lieut. M. C.-O., Aberdeen, Wash., Naval Reserve Armory, Aberdeen, Wash.
WEAVER, Harry S., Jr., Lieut. M. C.-O., Philadelphia, N. H., Philadelphia
WEBB, Charles E., Lieut. M. C.-V. (S.), El Paso, Texas, N. H., Parris Island, S. C.
WEBB, James S., Jr., Lieut. M. C.-V. (G.), New Orleans, N. R. Armory, New Orleans
WELSH, Clyde L., Lieut. M. C.-O., Seattle, 11th Bn., U. S. M. C. R., Seattle
WERTHMAN, Paul A., Lieut. (j. g.) M. C.-V. (G.), Gustine, Calif., N. H., San Diego, Calif.
WESTON, K. R., Lieut. M. C.-V. (S.), Allentown, Pa., N. H. Philadelphia
WHALEN John M., Lieut. Comdr. M. C.-O., Grand Rapids, Mich., N. H., San Diego, Calif.
WHEELER, Howard L., Lieut. Comdr. M. C.-V. (S.), Baltimore, N. R. S., Baltimore
WHITEHEAD, Hugh G., Jr., Lieut. (j. g.) M. C.-O., Baltimore, Norfolk Naval Hospital.
WIESINGER, Warren E., Lieut. (j. g.) M. C.-V. (G.) Oakland, Calif., N. H., San Diego, Calif.
WILDEBUSH, Frank F., Lieut. M. C.-V. (S.), Minneapolis, Naval Mobile Hospital No. 1
WILDMAN, Henry V., Lieut. Comdr. M. C.-O., New York, N. H., Pensacola, Fla.
WILLIAMS, George Z., Lieut. (j. g.) M. C.-O., Richmond, Va., Norfolk Naval Hospital
WILLIAMS, Harry D., Jr., Lieut. (j. g.) M. C.-V. (G.), San Francisco, N. R. A. B., Oakland, Calif.
WILLIS, Park Weed, Jr., Lieut. Comdr. M. C.-V. (S.), Seattle, 1st Bn., U. S. M. C. R., Seattle
WILSON, Leonard L., Lieut. Comdr. M. C.-V. (S.), Cheyenne, Wyo., N. H., Pensacola, Fla.
WIRIG, Marres H., Lieut. M. C.-O., Madison, Wis., Madison, Wis.
WITKIN, Leonard E., Lieut. (j. g.) M. C.-V. (G.), Brooklyn, N. R. A. B., Navy Yard, Philadelphia
YOUNG, Vincent T., Lieut. (j. g.) M. C.-O., Methuen, Mass., N. H., Chelsea, Mass.
ZOBEL, Jerome F., Lieut. (j. g.) M. C.-V. (G.), San Francisco, N. H., San Diego, Calif.

NEW MEDICAL OFFICERS

As the result of competitive examinations held in July, the following Reserve Officers have been appointed as first lieutenants in the Medical Corps of the Regular Army. Following the names are the home town and the medical school:

BARKER, Warren J., Checotah, Okla., Tulane.
BEDDOW, Leon D. Dallas, Texas, Baylor.
BEELER, Thomas T., Jr., Norman, Okla., University of Oklahoma.
BERRY, C. Z., Cincinnati, University of Cincinnati.
BORNSTEIN, Joseph H., Chelsea, Mass., Boston University.
BOZALIS, George S., St. Louis, University of Oklahoma.
BUTKUS, Walter A., Chicago Heights, Ill., Loyola.
CLEVELAND, William H., Rochester, Minn., Northwestern.
DICE, Wibur D., State University of Iowa.
DUBUY, Carl T., Tulia, Texas, University of Michigan.
FERRELL, Lee F., Memphis, Tenn., University of Tennessee.
FRESE, Frederick J., Jr., Barksdale Field, La., St. Louis University.
FURST, John N., Hallock, Minn., University of Minnesota.
GUNN, Edward M., Providence, R. I., Syracuse University.
HARNEY, John P., New York, Long Island University.
HARWOOD, Samuel C., Los Angeles, University of Southern California.
HUMPHREYS, James W., Jr., Cincinnati, Medical College of Virginia.
HURTEAU, William W., Washington, D. C., State University of Iowa.
JACKSON, William M., Dickson, Tenn., University of Tennessee.
LAWN, R. A., Minneapolis, University of Minnesota.
LONG, Irl R., Washington University.
LOOK, William B., Fort Wayne, Mich., Washington University.
MOODY, William M., Cincinnati, University of Cincinnati.
O'BRIAN, Spencer A., Chicago, University of Georgia.
PEARSON, Murble H., Dillon, Mont., University of Oklahoma.
PETTIT, Vernon D., Memphis, Tenn., University of Tennessee.
SCHMITZ, W. G., Monterey, Calif., Loyola University
SHUEY, H. E., Cottage Grove, Ore., University of Oregon.
SPANN, Franklin L., San Antonio, Texas, Baylor University.
SWIFT, Edward V., Austin, Texas, University of Texas.
TALBOTT, Charles H., St. Louis, Washington University.
TILL, Jacob R., Jr., Klawock, Alaska, Tulane University.
TOUSIGNANT, Harvey, G., Oconto, Wis., Marquette University.
WARD, David P., Pemberville, Ohio, Ohio State University.

CONTROL OF VENEREAL DISEASE

An agreement by the War and Navy departments, the Federal Security Agency and state health departments on measures for the control of venereal disease in areas in which armed forces or national defense employees are concentrated was recently made public by the U. S. Public Health Service. The agreement outlined services to be developed by state and local health and police authorities in cooperation with medical corps of the U. S. Army, the bureau of medicine and surgery of the U. S. Navy, the public health service and interested voluntary organizations. Early diagnosis and treatment were stressed, the military personnel to be under the supervision of the Army and Navy and civilian population under local health departments. It was agreed that, when authentic information could be obtained as to the probable source of infection of military or naval personnel, the facts would be reported by medical officers of the Army or Navy to state or local health authorities. Recalcitrant infected persons will be forcibly isolated during the period of communicability. Health authorities, both military and civilian, also agree to cooperate with police departments in decreasing as far as possible the opportunities for contacts with infected persons. Among other measures, an aggressive program of education regarding the dangers of venereal diseases was proposed. Finally the health and police authorities requested the assistance of social hygiene societies and other welfare organizations in developing and stimulating public support for the measures to be undertaken.

ORGANIZATION SECTION

OFFICIAL NOTES

SCIENTIFIC EXHIBIT

Application blanks are now available for space in the Scientific Exhibit at the Cleveland Session of the American Medical Association, June 2 to 6, 1941. They may be obtained by communicating with the Director, Scientific Exhibit, American Medical Association, 535 North Dearborn Street, Chicago.

APPLICATIONS TO THE COMMITTEE ON SCIENTIFIC RESEARCH FOR GRANTS

The Committee on Scientific Research of the American Medical Association invites applications for grants of money to aid in research on problems bearing more or less directly on clinical medicine. Preference is given to requests for moderate amounts to meet specific needs. Application forms may be obtained from the Committee, 535 North Dearborn Street, Chicago.

RADIO BROADCASTS

Doctors at Work is the title of the sixth annual series of dramatized radio programs being presented by the American Medical Association and the National Broadcasting Company.

The series opened Wednesday, November 13, and will run for thirty consecutive weeks, closing with a broadcast from the A. M. A. meeting at Cleveland on June 3, 1941. The program is scheduled for 10:30 p. m. eastern standard time (9:30 central, 8:30 mountain, 7:30 Pacific time) over the Blue network, other N. B. C. stations and Canadian stations.

These programs are broadcast on what is known in radio as a sustaining basis; that is, the time is furnished gratis by the radio network and local stations and no revenue is derived from the programs. Therefore, local stations may or may not take the programs, at their discretion, except those stations which are owned and operated by the National Broadcasting Company.

The programs will dramatize what modern medicine offers the individual in the way of opportunities for better health and the more successful treatment of disease. Incidental to this main theme the programs will explain the characteristics of the different fields of modern medicine and its specialties.

Descriptive posters for local distribution may be had gratis from the Bureau of Health Education, American Medical Association, 535 North Dearborn Street, Chicago. Program titles will be announced weekly in THE JOURNAL and monthly in *Hygeia*, The Health Magazine.

The next three programs to be broadcast, together with their dates and their topics, are as follows:

November 20, "The Years in Training."
November 27, "Through Hospital Corridors."
December 4, "Paging Doctor Drew."

NATIONAL BOARD MEETING IN CHICAGO

The board of directors of the Woman's Auxiliary to the American Medical Association will hold its regular meeting at the Palmer House, Chicago, beginning at 9 a. m., November 29.

WOMAN'S AUXILIARY

Colorado

The woman's auxiliary to the Montrose County Medical Society was organized in April and has had several interesting meetings with its nine members. In August the meeting was held in Olathe at the home of Mrs. R. R. Rigg, with Mrs. Richard Waldapfel, of Grand Junction, as guest speaker. She is an Austrian refugee in the United States with her husband, who is an otolaryngologist. Mrs. Waldapfel spoke on "Vienna Before and After Hitler." In August the auxiliary met at the home of Mrs. Lockwood for a mountain trout dinner, part of a limit catch made by the hostess, who is an expert angler. The September meeting was held at the home of Mrs. Norman A. Brethouwer, where arrangements were made to hold a venison dinner for the doctors, who were guests of the auxiliary at the October meeting. The primary function of the auxiliary is to sew for local hospitals.

The annual meeting of the woman's auxiliary to the Colorado State Medical Society was held at Glenwood Springs, September 12-14. Mrs. Lorenz Frank, of Denver, president of the auxiliary, presided. At the annual luncheon held Friday, September 13, with 101 members present, Mrs. V. Eugene Holcombe, of Charleston, W. Va., national president of the woman's auxiliary, gave the principal address.

New York

The woman's auxiliary to the Cayuga County Medical Society met, September 19, at the home of Mrs. George B. Adams. There were fifteen members present and ten women from Seneca County interested in forming an auxiliary.

Members of this county were busy during the summer cooperating in a sale held by the blind and working in the health booth during the county fair. A luncheon opened the first fall meeting of the auxiliary to the Broome County Medical Society at the Binghamton Club, October 10. Mrs. John H. Robertson, presiding, named committee heads for the ensuing year.

Mrs. L. H. Kice, state auxiliary president, was a guest at a well attended meeting of the county auxiliary in Saratoga Springs, October 2. "The standard of our auxiliary is 'service to humanity' and to march in its progress," was the keynote of her address.

Pennsylvania

The Franklin County auxiliary met in June at Chambersburg. At the roll call, members answered by giving a few facts on some pioneer in medicine. Mrs. John H. Doane, state auxiliary president, was guest speaker.

The annual charity party of the Lehigh County auxiliary was held in May with about 150 members and guests present. The proceeds are to be used toward the Medical Benevolence Fund. In June 100 members of the auxiliary enjoyed the annual "President's Party."

The annual meeting of the Montgomery County auxiliary was held in May. Mrs. John H. Doane, president of the state auxiliary, was guest speaker. She stated that the Pennsylvania state auxiliary holds the largest membership in the United States.

Washington

The annual meeting of the woman's auxiliary to the Washington State Medical Association was held in Tacoma in August with Mrs. V. E. Holcombe, national president, as guest of honor. Mrs. R. E. Mosiman of Seattle, president-elect of the national auxiliary, was also present. Mrs. C. E. Sears, of Oregon, president-elect of Oregon state auxiliary, who was instrumental in the organization of the Washington state auxiliary, was a special guest. Mrs. Luman S. Roach, of Kalama, completed a successful year as state president. Mrs. George E. Hoxsey, Wenatchee, took the chair as the president for 1940-1941 and told of plans for the coming year, stressing the importance of Red Cross work and subscriptions to the *Bulletin*.

Medical News

(PHYSICIANS WILL CONFER A FAVOR BY SENDING FOR THIS DEPARTMENT ITEMS OF NEWS OF MORE OR LESS GENERAL INTEREST: SUCH AS RELATE TO SOCIETY ACTIVITIES, NEW HOSPITALS, EDUCATION AND PUBLIC HEALTH.)

CALIFORNIA

Physicians Tour Vineyards. — Fifty-four physicians in northern California visited some of the historic vineyards and wine cellars of the Napa Valley, October 13, on a tour sponsored by the Society of Medical Friends of Wine. The society was organized in 1939 to stimulate scientific research on wine. Officers include Drs. Thomas F. Mullen, president; Marius A. Francoz, vice president; Randolph G. Flood, secretary, and Harry E. Alderson, treasurer. All are from San Francisco.

Symposium on Heart Disease. — The eleventh annual postgraduate symposium on heart disease of the San Francisco Heart Committee will be held November 26-28. Sessions will be alternated in the hospitals of Stanford University School of Medicine and the University of California Medical School and San Francisco Hospital. The course will include demonstrations of patients presenting problems in the various types of heart disease, discussion and evaluation of specific diagnostic procedures and therapy, differential diagnosis and treatment. There will be ward rounds and special classes in x-ray, fluoroscopy and electrocardiography. At a dinner session Wednesday evening, November 27, Chauncey D. Leake, Ph.D., will discuss "Harvey, the Heart, and War" and an arrangement by Dr. Harold H. Rosenblum of "The Heart Sounds Again" will be presented. A series of forum meetings on "Public Health and Community Aspects of Heart Disease" will be held throughout the day at Mount Zion Hospital.

DELAWARE

Society News.—A symposium on chest conditions was presented before the New Castle County Medical Society in Wilmington, October 15; the speakers were Drs. Gerald A. Beatty on "Spontaneous Pneumothorax"; Harold A. Tarrant, "Presentation of Lung Changes from an Unusual Cause," and Eugene P. Pendergrass, Philadelphia, "The Healthy Chest and the Modifying Influences of Silicosis."

FLORIDA

Regional Meetings. — The fourth annual meeting of the Southeast Medical District Society was held at Coral Gables, November 2, with the following speakers, among others:

Dr. Frederick H. Dieterich, Miami, Medicine and the Florida Criminal Law.
Dr. Elliott M. Hendricks, Fort Lauderdale, The Colon as a Focus of Infection.
Dr. Russell B. Carson, Fort Lauderdale, Pain Produced by Urologic Disease.
Dr. John R. Boling, Tampa, Vaginal Hysterectomy.

Speakers at the fourth annual meeting of the Southwest Medical District in Dunedin, October 31, included:

Dr. Alvin L. Mills, St. Petersburg, A Review of Some of the More Commonly Used Drugs in Urology.
Dr. David R. Kennedy, Sarasota, The Life of the Doctor.
Dr. Robert B. Harkness, Lake City, Occlusive Lesions of the Peripheral Blood Vessels.

At the fourth annual meeting of the South Central Medical District in Fort Pierce, November 1, the program was presented, among others, by:

Dr. Isaac M. Hay, Melbourne, Loss of Integrity of the Radio-Ulnar Joint in Colles' Fracture.
Dr. Duncan T. McEwan, Orlando, Perirectal Infection from the Anaerobic Streptococcus.
Dr. Joseph S. Stewart, Miami, Some Minor Surgical Procedures.

GEORGIA

Special Society Election. — Dr. Richard E. Newberry, Atlanta, was chosen president of the Georgia Industrial Surgeons Association, succeeding Dr. Robert L. Rhodes, Augusta, at its third annual meeting in Atlanta, September 25. Other officers include Drs. Benjamin H. Minchew, Waycross, vice president, and John W. Simmons, Brunswick, secretary-treasurer.

Matas Medal Awarded to Dr. Elkin.—The Matas Award in Vascular Surgery was presented, November 14, to Dr. Daniel Collier Elkin, Joseph Brown Whitehead professor of surgery at Emory University School of Medicine, Atlanta, in recognition of his work on heart wounds and aneurysms. The award was created by the Violet Hart Fund and is administered by the Tulane Educational Fund and a committee composed of Drs. Emile Bloch, Lucian H. Landry, Isidore Cohn and Edward William Alton Ochsner, New Orleans. Named in honor of Dr. Rudolph Matas, professor of general and clinical surgery emeritus, Tulane University of Louisiana School of Medicine, the medal is presented "to that North American surgeon who had contributed outstanding work in vascular surgery." The first presentation was made in 1934 to Dr. Mont R. Reid, Cincinnati, and the second in 1937 to Prof. Reynaldo dos Santos, Lisbon, Portugal. Born in Louisville, Ky., Dr. Elkin graduated at Emory University School of Medicine in 1920, joining its teaching staff three years later.

ILLINOIS

Regional Meeting of College of Physicians.—The sixth annual regional meeting of the American College of Physicians for Illinois (outside of Cook County) including members from Wisconsin, was held at Rockford, October 16. Participating were:

Dr. Ernest E. Irons, Chicago, Aspiration Pneumonia.
Dr. Elston L. Belknap, Milwaukee, The Responsibility of the Internist to the Field of Industrial Medicine.
Dr. John A. Schindler, Monroe, The Clinical Importance of Various "Symptomless" Anomalies of the Gastrointestinal Tract.
Dr. Carroll W. Osgood, Wauwatosa, Physical Aspects of Depressive Psychoses.

Personal. — Dr. Zoda D. Lumley, Wood River, has been named health officer of Roxana, succeeding the late Dr. Christian H. Diehl.——Dr. Louis H. Cohen, clinical director of the Manteno State Hospital, resigned, September 16, to accept a similar position at a state hospital in Norwich, Conn. He will be succeeded at Manteno by Dr. Louis B. Shapiro, Elgin. ——The Sangamon County Medical Society gave a dinner in honor of Dr. James A. Day, Springfield, October 3, to observe his completion of fifty years in the practice of medicine. He was made a member of the Fifty Year Club of the state medical society.

Chicago

Society News. — Dr. Oliver S. Ormsby will deliver the presidential address before the twenty-fifth annual meeting of the Institute of Medicine of Chicago, December 3, on "Pellagra: Special Reference to Etiology, Cutaneous Manifestations and Treatment." —— The Chicago Society of Internal Medicine will be addressed, November 25, by Drs. Fred W. Preston and Willard O. Thompson on "Persistence and Recurrence of Toxic Goiter Following Subtotal Thyroidectomy"; Raphael Isaacs, Ann Arbor, Mich., "Red Blood Cell Size as an Aid in Diagnosis, Prognosis and Treatment," and Italo F. Volini, Robert O. Levitt and Hugh B. O'Neil, "Sulfathiazole in the Treatment of Pneumococcus Pneumonia with a Comparative Study Utilizing Sulfapyridine Therapy" and "Cutaneous and Conjunctival Manifestations of Sulfathiazole Intoxication." —— The Chicago Society of Allergy was addressed, October 21, by Dr. Meyer R. Lichtenstein on "The Relation of Atopy to Immunology."——A symposium on the treatment of pneumonia will be presented before the Chicago Medical Society at the Chicago Woman's Club, November 20. Dr. Gerald S. Shibley, Cleveland, will discuss "Chemotherapy" and Dr. Italo F. Volini, "Special Serum Therapy."

KENTUCKY

District Meetings.—A symposium on "Immediate Management of Emergencies" formed the program of a meeting of the sixth and seventh councilor districts of the Kentucky State Medical Association at Somerset, October 22, with the Pulaski County Medical Society as host. The speakers were Drs. Emmet F. Horine, Louisville, on "Cardiac Emergencies: Their Diagnosis and Treatment"; Robert F. Monroe, Louisville, "Postpartum Hemorrhage"; Alexander J. Alexander, Lexington, "Convulsions in Children: Differential Diagnosis and Emergency Treatment"; Augustine Thornton Scott, Lexington, "Coma: Differential Diagnosis and Emergency Treatment," and William D. Reddish, Lexington, "Automobile Accident Injuries: Their Immediate Management."——The Shelby County Medical Society was host to the fifth councilor district meeting at Shelbyville, October 17. The speakers were Drs. William S. Snyder Jr., Frankfort, on "Importance of Early Treatment for Squint"; Marinus H. Pulskamp, Louisville, "Importance of Anorectal Examination," and Emmet F. Horine, Louisville, "Indications and Contraindications for the Use of Digitalis." In the afternoon Drs. Philip F. Barbour and James H. Pritchett, Louisville, conducted a pediatric clinic.

MASSACHUSETTS

Monographs in Medicine and Public Health. — To encourage the publication of books and monographs of scientific importance by members of the Harvard Medical School and the Harvard School of Public Health, a sum of money has been provided to be used for a period of three years as a revolving publication fund. An editorial committee has been appointed to administer this fund and to select books and monographs to be included in a series entitled Harvard University Monographs in Medicine and Public Health. Members of the committee are Albert Baird Hastings, Ph.D., chairman; Drs. Walter B. Cannon, James Howard Means, Simeon Burt Wolbach and Katherine R. Drinker, executive secretary. Members of the faculties of the medical school and the school of public health who have written or who contemplate writing books or monographs suitable for such a series are invited to consult with the editorial committee and to submit their manuscripts for inclusion in the series. If financial assistance for publication of a book is required, the committee is authorized after arrangement with the Harvard University Press to use the publication fund for this purpose. To further the economical manufacture and the successful sale of books in the monograph series, the editorial committee is prepared to supply to authors, through its executive secretary, consulting advice on the proper preparation of manuscripts for publication and to make sure that announcements of books published under its auspices are widely circulated to especially interested groups. Number 1 of the Harvard University Monographs in Medicine and Public Health, to be published this month by the Harvard University Press, will be "The Endocrine Function of Iodine" by Dr. William T. Salter. Additional titles, now in prospect, will be announced later.

NEBRASKA

Personal.—Dr. David C. Hilton, Lincoln, a colonel in the Nebraska National Guard, has been placed on the inactive list with the rank of brigadier general. Dr. Hilton requested the change of status, as he is nearing the retirement age, according to the *Nebraska State Medical Journal.* He helped to organize the 110th Medical Regiment of the state unit in 1923. At that time he was commissioned a lieutenant colonel and later was promoted to a full colonelcy and placed in command of the regiment.

District Meeting. — The annual meeting of the Seventh Councilor District Medical Society was held in Geneva, October 17. Scientific speakers, all of Omaha, were Drs. Esley J. Kirk on "Correlation of the Physiological and Chemical Changes in Nephritis and Their Relation to Treatment"; John Harry Murphy, "Poliomyelitis—Diagnosis and Treatment," and Maine C. Andersen, "Practical Application of Heart Tracings." Drs. Clayton F. Andrews, Lincoln, and William P. Wherry, Omaha, president and president-elect of the Nebraska State Medical Association, spoke at an evening session.

NEW JERSEY

The Martland Lecture.—The sixth annual Harrison S. Martland Lecture, sponsored by the Essex County Anatomical and Pathological Society, will be presented at the Academy of Medicine of Northern New Jersey, Newark, November 29, by Dr. William Boyd, professor of pathology and bacteriology, University of Toronto Faculty of Medicine. Dr. Boyd's subject will be "Recent Increase in Bronchial Carcinoma."

Annual Clinical Conference. — The Medical Society of New Jersey will hold its third annual clinical conference in Essex County hospitals, November 27-28, on the general theme of "The General Practitioner." There will be one program of scientific papers at the Academy of Medicine of Northern New Jersey, Newark, Wednesday afternoon, November 27, with the following speakers, all of Newark:

Dr. Harrison S. Martland, The Pathologist and the General Practitioner.
Dr. Henry H. Kessler, Evaluation of Disability in Industrial Accidents.
Dr. Elmer G. Wherry, Prematurity.
Dr. William L. James, The Sulfanilamide Group in Treatment of Gonorrhea.

The annual dinner will be held Wednesday evening at the Newark Athletic Club, with Dr. William J. Carrington, Atlantic City, as toastmaster and Harold Major, D.D., as the principal speaker, on "The Future of America." Drs. Harry N. Comando, Newark, president of the Essex County Medical Society, and Watson B. Morris, Springfield, president of the Medical Society of New Jersey, will also speak.

NEW YORK

Annual Health Supplement.—The Third Annual Health Supplement, prepared in cooperation with the Medical Society of the County of Nassau, was published, October 29, by the *Nassau Daily Review-Star,* Rockville Centre, New York. Consisting of forty pages in tabloid form, the supplement is devoted chiefly to the various aspects of medical preparedness. It includes articles outlining the preparedness activities of the Medical Society of the State of New York, the Nassau County society and the American Medical Association. In an editorial entitled "Health—for Preparedness," the paper says:

> Last year our Health Supplement urged people to care for their health because they owed it to themselves. This year we are beginning to see that good health is something our people owe not only to themselves but also to their country. In the days to come the worker in our factory, the civil servant, the man who handles our transportation and our communication, yes, and the man and woman who work to "keep the home fires burning" will be just as important in the common cause as the pilot of a bomber, the man who directs an anti-aircraft gun or the soldier who digs a trench. It mustn't be said that America failed because her people neglected the priceless heritage of health.

New York City

Fellowship Available for Latin-American Physician.—Mount Sinai Hospital announces that a fellowship has been established to enable physicians from the Latin-American countries to receive graduate training at this hospital. The Dazian Foundation for Medical Research has provided a grant of a sum up to $2,000 for the stipend, which will include the cost of travel. The holder will be known as the Dazian Fellow. The fellow may arrange to serve in any of the clinical or laboratory departments of the hospital, the choice depending on the needs and wishes of the physician selected for the position. Application forms may be obtained by addressing the director of the hospital, Dr. Joseph Turner, Mount Sinai Hospital, Fifth Avenue and One Hundredth Street, New York. Qualifications of applicants will be reviewed by the hospital's committee on fellowships and the candidate will be notified in due time.

Medal and Cancer Poster Prizes Awarded.—Dr. James Ewing, director emeritus and consulting pathologist of Memorial Hospital for the Treatment of Cancer and Allied Diseases, received the Clement Cleveland Medal of the New York City Cancer Committee at its fourteenth annual dinner, October 30. At the same time prizes were awarded in the poster contest conducted by the National Alliance of Art and Industry for the American Society for the Control of Cancer. The first prize of $1,000 went to a young Viennese refugee artist, Henry Koerner. Mrs. Robert G. Mead, who established the medal award in 1937 in memory of her father, the late Dr. Cleveland, made the presentation to Dr. Ewing, who responded with an address. Rear Admiral Ralph Whitman (C. E. C.), U. S. Navy, made an address on "Health for Defense." Clarence C. Little, Sc.D., managing director of the American Society for the Control of Cancer, presented the prizes for the poster contest. In addition to the first prize to Mr. Koerner, prizes of $500, $250, $50, $25 and five of $10 each were awarded.

OKLAHOMA

Society News.—Dr. Otto Jason Dixon, Kansas City, Mo., will address the Tulsa County Medical Society, Tulsa, November 25, on "An Operation for the Correction of Deafness." ——Dr. John M. Carson, Shawnee, discussed common rectal lesions at a meeting of the Pottawatomie County Medical Society, October 19, in Shawnee.——Dr. Paul B. Champlin, Enid, addressed the Garfield County Medical Society, Enid, October 24, on "Papillary Cystadenoma of the Ovary" and Drs. Frank T. Joyce and Charles J. Roberts discussed electrocardiography.

Public Health Association Meeting.—The annual meeting of the Oklahoma Public Health Association was held in Tulsa, October 3-4. The speakers included Clair E. Turner, Dr.P.H., professor of public health education at Massachusetts Institute of Technology, Cambridge, on "The Trend of School Health Education"; Miss Katherine Lenroot, chief of the Children's Bureau, Washington, D. C., "The Future of the Public Health Worker" and Dr. Henry H. Turner, Oklahoma City, president of the Oklahoma State Medical Association, "Public Health, A New Essential to Organized Medicine." Mr. Burley Walker of the staff of the state department of health was elected chairman.

OREGON

Annual Registration Due December 1.—All practitioners of medicine and surgery holding licenses to practice in Oregon are required by law to register annually on or before December 1 with the secretary of the state board of medical examiners and at that time to pay a fee of $5. A practitioner failing to register is subject to a penalty of $1 for each thirty days, or part thereof, of default and his failure to register within ninety days after December 1 is a misdemeanor.

PENNSYLVANIA

Society News. — Dr. William D. Whitehead, Scranton, addressed the Montour County Medical Society, Danville, October 18, on "Contact Dermatitis."——Dr. Edward L. Bauer, Philadelphia, addressed the Delaware County Medical Society, Chester, October 10, on "An Immunizing Program Against Communicable Diseases in Childhood for the Family Physician."——Dr. Claude Merrill Leister, Bethlehem, addressed the Northampton County Medical Society, October 18, on "Observations on Children's Blood."——Dr. John P. Henry, Pittsburgh, addressed the Washington County Medical Society, Washington, October 9, on "Diseases of the Peripheral Arteries and Veins."——Dr. Ralph R. Mellon, Pittsburgh, addressed the Westmoreland County Medical Society, Greensburg, October 15, on "Physiologic and Therapeutic Consideration of Sulfanilamide, Sulfapyridine and Sulfathiazole."

Pittsburgh

Fifth Renziehausen Lecture. — Dr. Cyril N. H. Long, Sterling professor of physiological chemistry, Yale University School of Medicine, New Haven, Conn., will deliver the fifth Renziehausen Memorial Lecture at the meeting of the Allegheny County Medical Society, November 19. His subject will be "The Endocrine Control of Metabolism."

PUERTO RICO

Inter-American Hospital Institute. — The first Inter-American Institute of Hospital Administrators will be held in San Juan, December 1-14, with headquarters at the Hotel Condado and sessions at the School of Tropical Medicine of the University of Puerto Rico. Invitations have been sent to hospital administrators and assistant administrators and heads of administrative departments throughout the Americas. Among those who will lecture are:

Dr. Eduardo Garrido Morales, San Juan, commissioner of health of Puerto Rico.
Dr. Malcolm T. MacEachern, associate director, American College of Surgeons, Chicago.
Dr. Leopoldo Figueroa Carreras, San Juan, president of the Territorial Charities Board of Puerto Rico.
Dr. Bert W. Caldwell, executive secretary, American Hospital Association, Chicago.
Dr. Arthur C. Bachmeyer, director of clinics, University of Chicago.
Julio B. Ortiz, Ph.D., dean, College of Business Administration, University of Puerto Rico.
Mr. James A. Hamilton, director of the New Haven Hospital, New Haven, Conn.
Mr. Félix Lamela, administrator, University Hospital, San Juan.
Dr. José Rodriguez Pastor, chief, bureau of tuberculosis, Department of Health, San Juan.
Dr. Ramón Señeriz, director, Bayamón District Hospital.

The institute is sponsored by the American College of Hospital Administrators and the American Hospital Association, in cooperation with the University of Puerto Rico and its School of Tropical Medicine; the Insular Department of Health, the Puerto Rico Medical Association, the Territorial Charities Board and the Puerto Rico Hospital Council.

GENERAL

Academy of Pediatrics.—The tenth annual meeting of the American Academy of Pediatrics will be held at the Hotel Peabody, Memphis, Tenn., November 18-20. Dr. Edwin E. Osgood, Portland, Ore., will open the scientific program with a talk on "Chemotherapy of Staphylococcus Infections." A symposium on virus diseases will then be presented by Fred D. Stimpert, Ph.D., Detroit; Dr. Ernest W. Goodpasture, Nashville, Tenn., and Dr. Joseph Stokes Jr., Philadelphia. Round table discussions will be devoted to the following subjects: anemias of infancy; appraisal of quarantine requirements; causes and treatment of behavior problems in children; diarrhea and dysentery; infant feeding; intestinal parasites; malaria; methods of improving pediatric practice; neonatal asphyxia; what's wrong with my child's feet; adiposogenital dystrophy; allergy; chemotherapy; treatment of pneumonia in children; vitamins from the standpoint of the practicing pediatrician, and methods of improving pediatric practice.

Southwestern Medical Association.—The annual meeting and clinical conference of the Southwestern Medical Association will be held in Tucson, Ariz., November 21-23, under the presidency of Dr. Orville E. Egbert, El Paso, Texas. Headquarters will be at the Pioneer Hotel. Guest speakers who will give several addresses each at general assemblies and section meetings are:

Dr. George E. Fahr, professor of internal medicine, University of Minnesota Medical School, Minneapolis.
Dr. Richard B. Cattell, Lahey Clinic, Boston.
Dr. Thomas T. Mackie, assistant clinical professor of medicine, Columbia University College of Physicians and Surgeons, New York.
Dr. John S. Lundy, section on anesthesia, Mayo Clinic, Rochester, Minn.
Dr. Willard M. Allen, professor of obstetrics and gynecology, Washington University School of Medicine, St. Louis.
Dr. Carleton Mathewson Jr., associate professor of surgery, Stanford University School of Medicine, San Francisco.
Dr. William L. Benedict, professor of ophthalmology, University of Minnesota Graduate School of Medicine, Mayo Foundation, Rochester.

There will also be a meeting of the Southwestern Academy of Eye, Ear, Nose and Throat with Dr. Benedict and Dr. Horace G. Merrill, San Diego, Calif., as guest speakers.

Specialty Board Examinations.—The American Board of Obstetrics and Gynecology announces that a written examination and review of case histories (Part I) for Group B candidates will be held in various cities of the United States and Canada Jan. 4, 1941. Notice of the place will be sent to each candidate in advance. Candidates who complete this examination proceed to the Part II examination to be held for all candidates in Cleveland, May 28 to June 1, 1941, immediately preceding the annual session of the American Medical Association. Applications for admission to Group A (Part II) examinations must be on file in the secretary's office not later than March 15. After January 1942 there will be only one classification of candidates and all will be required to take the Part I and Part II examinations. The board announces a change in the case record ruling, effective Jan. 1, 1942. This ruling should now read "It is preferable that the number of cases submitted should not be more than half (25) of the total number of fifty (50) cases required." For further information and application blanks, address Dr. Paul Titus, secretary, 1015 Highland Building, Pittsburgh.

Fellowships from the Lalor Foundation.—Six fellowship awards in chemistry will be granted by the Lalor Foundation for the academic year 1941-1942, four postdoctorate awards of $2,000 each and two awards of $2,500 each. The recipients must have the equivalent of a Ph.D. degree and the applicants will be judged by their previous training, demonstrated competence and promise in their special fields. The selection of fellows has been assigned to six chemists, each of whom will choose the holder of the award for his institution. The judges are: Roger Adams, Ph.D., University of Illinois, Urbana; Hans Clarke, D.Sc., Columbia University, New York; Frederick G. Keyes, Sc.D., Massachusetts Institute of Technology, Cambridge; Charles A. Kraus, Ph.D., Brown University, Providence, R. I.; Arthur B. Lamb, Ph.D., Harvard University, Cambridge, and Dr. Eli Kennerly Marshall Jr., Johns Hopkins University School of Medicine, Baltimore. Detailed information and application forms may be obtained by addressing Charles Lalor Burdick, Ph.D., secretary of the Lalor Foundation, Wilmington, Del., or by direct communication with one of the chemists named. Applications should be filed before December 31.

Government Services

Committees to Consider Industrial Health

The U. S. Department of Labor has announced the appointment of four committees to consider accident, health, compensation and housing problems in the tri-state mining district of Missouri, Kansas and Oklahoma. According to a release from the department, this is a leading area in the mining of zinc and lead. The committees will work out a joint program for remedial action for the better control of work hazards, particularly in reference to silicosis exposure and for improvement in the living conditions of miners. Jeff A. Robertson, commissioner, Kansas Department of Labor, is chairman of the committee on mine hazard control; Dr. Fred P. Helm, Topeka, secretary and executive health officer, Kansas State Board of Health, chairman of the committee on social and health problems; Associate Justice William A. Smith, Supreme Court of Kansas, chairman of the committee on workmen's compensation, and John A. Robinson, Miami, Okla., chairman of the committee on housing. V. A. Zimmer, U. S. Department of Labor, is the coordinating and liaison officer.

Foreign Letters

BUENOS AIRES

(From Our Regular Correspondent)

Oct. 11, 1940.

Control of Venereal Diseases in Argentina

The report of Dr. José J. Puente, in charge of the division of venereal disease and leprosy control of the national health department in Buenos Aires, presented at the eighth American Scientific Congress in Washington last May, has been published. Argentine legislation dealing with social diseases has been in force for about three years. According to the law, every hospital is to be equipped to treat venereal diseases. This provision has now largely been put into effect except in a few institutions for the chronic ill such as those for tuberculosis and leprosy. There are at present 709 antivenereal "servicios" in a total of 873 hospitals, representing an increase of 139 since the law went into effect. Calculated on population density, this represents one antivenereal welfare service for every 18,000 inhabitants. Most venereal diseases were found in certain regions in the northern part of the country, observations which are in accord with army statistics. Under the law every organization with fifty or more employees is compelled to maintain, without cost to the worker, a center for antivenereal prophylaxis and treatments. Where more than 100 workers are employed, the installation of a resident physician may be demanded. All large commercial and industrial concerns have organized a medical service of this kind. However, the law has not been in all cases rigorously enforced.

Prompt initial treatment and long continued subsequent attention have proved their worth. Systematic examinations for syphilis have not yet been undertaken in such a way as to determine the advantages of the procedure, but it is the general impression of physicians that more serious secondary effects, relapses and delayed manifestations of visceral and nervous phenomena may thereby be prevented. In gonorrhea, sulfonamide derivatives have more recently been employed. In chancroid the dmelcos type vaccines are used; in some cases sulfonamide derivatives have also been tried. Lymphogranulomatosis is treated with antimony preparations, antigen injections and sulfonamide derivatives. Favorable results have been obtained with antimony and surgical excision in a few cases of venereal granuloma.

TABLE 1.—*Primary and Secondary Syphilitic Infections*

	1934	1935	1936	1937	1938	1939
University clinic for venereal diseases in Buenos Aires (only fresh cases)...	104	96	87	67	52	32
Three dispensaries in the harbor area, Buenos Aires.....	480	497	513	338	234	193
Nineteen public aid dispensaries, Buenos Aires.....	1,060	1,636	1,333	1,141	834	319
Nine dispensaries in interior of country.....	1,765	1,653	1,069	1204	881	756

Complete statistics for venereal morbidity are not available. The figures given in table 1, however, indicate the reduction in new infections.

Since the law governing the control of venereal disease went into effect in May 1937, a significant, almost parallel, downward trend has been noted in all four groups (table 2). Accordingly, the incidence of chancroid and gonorrhea is lower, at least in Buenos Aires. Venereal lymphogranulomatosis, which had mounted during 1932-1937, seems to have lost its intensity. The majority of cases were found in the hospitals of Buenos Aires, the large cities of the interior, the harbor areas and the subtropical regions.

The significant recession in venereal morbidity is attributed to the intensified antivenereal campaign, the abolition of prostitution and the greater interest in personal prophylaxis. Army observations of recruits reflected in the subjoined figures confirm this: 1935, 1.11 per cent; 1936, 0.90 per cent; 1937, 0.86 per cent; 1938, 0.60 per cent; 1939, 0.65 per cent, and 1940, 49 per cent.

The explanation of the correlation between the abolition of prostitution and the decline of venereal diseases is sought in reduced promiscuity of sexual intercourse and, in consequence, in fewer opportunities for infection. The social effects of the abolition of prostitution were fiercely debated at the time the law was shaped. Statistics, however, for Buenos Aires do not confirm the fears entertained. Lack of funds has prevented as thorough an investigation of the sources of infection as is desirable, but wherever this could be done the favorable effects became promptly manifest.

TABLE 2.—*Downward Trend in Chancroid and Acute Gonorrhea*

	1934	1935	1936	1937	1938	1939
Incidence of Chancroid						
University clinic.....	19	108	47	11	8	5
Harbor area (three dispensaries).....	114	229	131	62	34	8
Public aid (nineteen dispensaries).....	704	1,098	616	150	36	17
Nine dispensaries in interior.....	141	280	349	258	34	46
Acute Gonorrhea						
University clinic.....	No report					
Harbor area (three dispensaries).....	709	853	912	683	763	610
Public aid (nineteen dispensaries).....	6,993	6,014	5,111	4,470	3,676	3,339
Nine dispensaries in interior.....	1,331	1,526	1,625	1,574	1,497	1,434

Higher Institute of University Culture

A bill has been introduced in the Argentine chamber of deputies for the founding of an institute that is to disseminate theoretical and practical information, especially for the benefit of physicians and to enable them to keep abreast of medical progress in the various branches of medicine in areas of the country where no university facilities are available. The institute is to be founded in Rosario, the second largest city of Argentina, under the control of the faculties of medicine of the country. The teaching personnel is to be drawn from the university staff and from physicians of scientific ability. The salary is to total 1,500 Argentine pesos (about $450) per month. The annual budget for the institute is set at 150,000 pesos (about $45,000). The underlying purpose of the plan is to afford postgraduate training to physicians who graduate from medical schools to practice in the interior. The demand on the isolated physician of the interior for a knowledge of special technics is greater than on his urban colleague. The institute is also to serve as a center of cooperation among physicians for the study of epidemics and their control. This again might serve to induce more physicians to practice in the interior and thus to aid the distribution of medical services away from the cities.

Marriages

KEITH WILLIAM MCFATRIDGE, Roxton, Texas, to Miss Margaret Daniel of Tuscaloosa, Ala., in September.

PETER CHARLES GAILLARD JR., Eutawville, S. C., to Miss Deane D. Shumate in Charleston, July 19.

JOHN R. CORKERY JR., Spokane, Wash., to Miss Margery Bordelon of Cottonport, La., in August.

JAMES CLAY WREN, Siler City, N. C., to Miss Edna Lynette Decker of Haworth, N. J., recently.

ROBERT W. SCHNEIDER, Cleveland, to Miss Marjorie Ann Rollin of Milwaukee in July.

FRANK HARMS, Aberdeen, Wash., to Miss Lois Kleiwer of Newport, September 7.

HARRY D. PASS to Miss Helen Thal, both of Seattle, August 20.

Deaths

Arthur Caradoc Morgan ✠ Philadelphia; Medico-Chirurgical College of Philadelphia, 1897; member of the House of Delegates of the American Medical Association in 1911 and from 1929 to 1938; became a member of the faculty at his alma mater in 1898 and served until the school was taken over in 1916 by the University of Pennsylvania to be developed as a graduate school; in the new school was associate professor of medicine from 1916 to 1922 and for several years was associate in medicine in the undergraduate department of the university; in 1922 went to Temple University School of Medicine as professor of applied therapeutics, remaining in that chair till 1928; emeritus professor of clinical medicine since 1930; fellow of the American College of Physicians; was president of the Philadelphia County Medical Society and the Medical Society of the State of Pennsylvania; formerly member of the state board of medical education and licensure; hospital affiliations included the Frankford Hospital, of which he was physician in chief and medical director for eight years, and the Medico-Chirurgical, Polyclinic, Samaritan and Garretson hospitals, where he had been at various times visiting physician; attending physician to the tuberculosis department of the Philadelphia General Hospital for many years; on the staff of the Eastern State Penitentiary Hospital; served during the World War; associate editor of the *Pennsylvania Medical Journal*; aged 70; died, October 21, in the Temple University Hospital of coronary occlusion.

Edward Everett Cornwall ✠ Brooklyn; College of Physicians and Surgeons, medical department of Columbia College, New York, 1890; one of the incorporators, ex-councilor and fellow of the American College of Physicians; member of the House of Delegates of the American Medical Association, 1911-1912; attending emeritus and consulting physician, Norwegian Lutheran Deaconesses' Home and Hospital; consulting physician, St. John's, Bethany Deaconess and Brooklyn Eye and Ear hospitals, Brooklyn, and the Southside Hospital, Bay Shore; author of "Clinical Treatise on Diseases of the Heart"; aged 74; died, October 6, of coronary thrombosis.

Louis P. H. Bahrenburg ✠ Medical Director, United States Public Health Service, Cleveland Heights, Ohio; Western Reserve University Medical Department, Cleveland, 1896; fellow of the American College of Surgeons; was commissioned in the United States Public Health Service as an assistant surgeon in 1900; subsequently was promoted through the various grades to the grade of medical director (ranking as colonel) in 1930 and was retired for age on Dec. 31, 1937; aged 66; died in October at Pemberville, Ohio.

Frederick William Zimmer, Rochester, N. Y.; University of Pennsylvania Department of Medicine, Philadelphia, 1882; member of the Medical Society of the State of New York; fellow of the American College of Surgeons; for many years medical consultant to the city school board; consulting surgeon, Rochester General, Rochester State, Park Avenue and Genesee hospitals; aged 82; died, September 21, of chronic cardiovascular renal disease.

Herbert Leroy Odell ✠ Sharon Springs, N. Y.; Albany Medical College, 1883; secretary and past president of the Schoharie County Medical Society; past president and secretary of the Schoharie County Tuberculosis and Public Health Association; for many years president of the school board and health officer of the village of Sharon Springs; aged 81; died, September 27, of arteriosclerosis.

Charles Browning Williams, Philippi, W. Va.; University of Virginia Department of Medicine, Charlottesville, 1895; member of the West Virginia State Medical Association; served during the World War; formerly bank president; on the staffs of the Myers Clinic Hospital, Philippi, and the Union Protestant Hospital, Clarksburg; aged 68; died, September 9, of pulmonary tuberculosis.

Alfred Joseph Helton, Yakima, Wash.; Rush Medical College, Chicago, 1902; member of the Washington State Medical Association and the American Association for the Surgery of Trauma; fellow of the American College of Surgeons; served during the World War; surgeon to St. Elizabeth's Hospital; aged 63; died, September 25, of cerebral hemorrhage.

Rudolph John Horvath, New York; Magyar Királyi Pázmány Petrus Tudományegyetem Orvosi Fakultasa, Budapest, 1912; member of the Medical Society of the State of New York; served during the World War; on the staff of the Beekman Hospital and the Mount Sinai Hospital; aged 54; died, September 28, of coronary thrombosis.

Lillian Ethel Taylor, Carmel, Calif.; University of Toronto Faculty of Medicine, Toronto, Ont., Canada, 1906; member of the American Academy of Ophthalmology and Otolaryngology; formerly on the staff of the Women and Children's Hospital, Chicago; died, September 26, of coronary thrombosis.

Richard Andrew Lawrence, Albany, N. Y.; Albany Medical College, 1907; member of the Medical Society of the State of New York; on the staffs of the Brady Maternity Home and St. Peter's Hospital; aged 58; died, September 28, of dissecting aneurysm of the thoracic and abdominal aorta.

Pleasant Leonidas Moon, Atlanta, Ga.; Atlanta Medical College, 1897; member of the Medical Association of Georgia; on the staffs of the Georgia Baptist Hospital and the Crawford W. Long Memorial Hospital; aged 71; died, September 22, in Pierson, Fla., of coronary thrombosis.

Sidney Solomon Oppenheimer ✠ Spokane, Wash.; McGill University Faculty of Medicine, Montreal, Que., Canada, 1898; fellow of the American College of Surgeons; aged 67; for many years on the staff of the Sacred Heart Hospital, where he died, September 20, of cerebral thrombosis.

John Irwin Gaston, Shawnee, Okla.; Chattanooga (Tenn.) Medical College, 1905; member of the Oklahoma State Medical Association; past president of the Pottawatomie County Medical Society; formerly county health officer; aged 63; died, September 24, of coronary thrombosis.

Eugene Baker, Ithaca, N. Y.; University of Michigan Department of Medicine and Surgery, Ann Arbor, 1882; member of the Medical Society of the State of New York; past president of the Tompkins County Medical Society; aged 87; died, October 14, of arteriosclerosis.

Nicholas C. Baumann, Highland, Ill.; Eclectic Medical Institute, Cincinnati, 1904; member of the Illinois State Medical Society; past president of the Madison County Medical Society; on the staff of St. Joseph Hospital; aged 64; died, October 13, of cerebral hemorrhage.

George H. Bolling, Pittsburgh; Howard University College of Medicine, Washington, D. C., 1904; member of the Medical Society of the State of Pennsylvania; aged 68; died, October 3, in the Western Pennsylvania Hospital of carcinoma of the esophagus.

Charles Burton Wormelle, Boston; Harvard Medical School, Boston, 1898; member of the Massachusetts Medical Society; formerly on the staffs of the City Hospital and the Children's Hospital; aged 68; died, September 16, of carcinoma of the stomach.

Albert Monroe Sittler, Bowmanstown, Pa.; Jefferson Medical College of Philadelphia, 1886; member of the Medical Society of the State of Pennsylvania; aged 76; died, October 2, in the Palmerton (Pa.) Hospital of cerebral embolism and pneumonia.

David F. Thompson, Toledo, Ohio; College of Physicians and Surgeons, medical department of Columbia College, New York, 1869; aged 93; died, September 20, in St. Vincent's Hospital of a fracture of the hip due to a fall and bronchopneumonia.

Robinson Leroy Bidwell ✠ Toledo, Ohio; Starling-Ohio Medical College, Columbus, 1908; fellow of the American College of Surgeons; surgeon, St. Vincent's and Women's and Children's hospitals; aged 56; died, September 26, of coronary sclerosis.

J. A. Moyers, Franklin, W. Va.; St. Louis College of Physicians and Surgeons, 1899; member of the West Virginia State Medical Association; aged 68; died, September 1, at a hospital in Harrisonburg, Va., of inoperable carcinoma of the stomach.

Joseph Daniel Slack ✠ New York; Columbia University College of Physicians and Surgeons, New York, 1904; on the staff of the Union Hospital; aged 59; died, September 7, in St. Luke's Hospital, New Bedford, Mass., of bronchopneumonia.

Martin Barbour Williams, Centerville, Ala.; Birmingham Medical College, 1907; member of the Medical Association of the State of Alabama; secretary of the Bibb County Medical Society; aged 62; died, September 28, of cerebral hemorrhage.

John Edward Di Giglia ✠ Lake Charles, La.; Tulane University of Louisiana School of Medicine, New Orleans, 1934; aged 30; on the staff of St. Patrick Hospital, where he died, September 22, of empyema and lung abscess.

Joseph Curry Allgood, Summerfield, La.; Louisville (Ky.) Medical College, 1894; member of the Louisiana State Medical Society; past president of the Claiborne Parish Medical Society; aged 69; died, September 27, of coronary thrombosis.

George Francis Rogan, Medina, N. Y.; Albany Medical College, 1893; member of the Medical Society of the State of New York; on the staff of the Medina Memorial Hospital; aged 70; died, October 2, of cerebral thrombosis.

Benjamin Escoe, New York; New York Homeopathic Medical College and Flower Hospital, New York, 1929; aged 39; died, September 22, in the Mount Sinai Hospital of hemolytic anemia following sulfapyridine therapy.

Thomas Freeman Howell, Alamosa, Colo.; University of Kansas School of Medicine, Kansas City, 1906; member of the Colorado State Medical Society; aged 68; died, September 24, in Salida of carcinoma of the prostate.

Ward Clifton Zeller, Visalia, Calif.; Ohio Medical University, Columbus, 1897; member of the California Medical Association; served during the World War; aged 66; died, September 26, of chronic myocarditis.

Michael James Rowe, Bridgeport, Conn.; College of Physicians and Surgeons, Baltimore, 1896; veteran of the Spanish-American War; aged 73; died, September 30, of cystitis, pyelitis and bladder and kidney stones.

John Ollie Hudson, Braman, Okla.; University of Nashville (Tenn.) Medical Deparment, 1910; member of the Oklahoma State Medical Association; aged 54; died, September 27, in Blackwell of cardiorenal disease.

George Brinton Woods, Curllsville, Pa.; College of Physicians and Surgeons, Baltimore, 1887; member of the Medical Society of the State of Pennsylvania; aged 77; died, September 29, of cerebral hemorrhage.

Joseph Michael Hilger, Iona, Minn.; College of Physicians and Surgeons of Chicago, School of Medicine of the University of Illinois, 1903; aged 60; died, September 4, of cerebral hemorrhage and hypertension.

Henry Clay Jarvis, Schell City, Mo.; Bennett College of Eclectic Medicine and Surgery, Chicago, 1882; University Medical College of Kansas City, Mo., 1901; aged 91; died, September 29, of coronary sclerosis.

Harry Preston Gibson, Leesburg, Va.; University of the South Medical Department, Sewanee, Tenn., 1909; member of the Medical Society of Virginia; aged 55; died, October 2, of cirrhosis of the liver.

Reginald Gilbert Bray, Niagara Falls, N. Y.; University of Vermont College of Medicine, Burlington, 1895; aged 81; died, October 5, of hemorrhage and ruptured aneurysm of the abdominal aorta.

Jacobum Phillippe Roulier, Salem, Mass.; School of Medicine and Surgery of Montreal, Que., Canada, 1888; aged 79; died, August 9, in the Beverly (Mass.) Hospital of cerebral hemorrhage.

Henry Ledbetter Flake, Leland, Miss.; Kentucky School of Medicine, Louisville, 1889; aged 73; died, September 29, in the King's Daughters Hospital, Greenville, of carcinoma of the stomach.

James Washington Benton, Peniel, Texas; Kentucky School of Medicine, Louisville, 1906; member of the State Medical Association of Texas; aged 79; died, September 29, of uremia.

George Henry Rawson, Washington, D. C.; George Washington University School of Medicine, Washington, 1918; aged 57; died, October 2, in Arlington, Va., of cardiac infarction.

Edwin Nichols Jr., Scottsbluff, Neb.; Starling Medical College, Columbus, 1894; aged 72; died, September 20, in the West Nebraska Methodist Episcopal Hospital of pneumonia.

Robert Lorain Smith, Syracuse, N. Y.; University of the City of New York Medical Department, 1892; aged 68; died, August 16, in Farmingdale, N. J., of coronary thrombosis.

George Massalon Murray ✠ Atlanta, Ga.; University of Virginia Department of Medicine, Charlottesville, 1909; aged 66; died, September 14, of myocarditis and influenza.

Clarke Sherwood Smith, Oakland, Calif.; University of Minnesota College of Medicine and Surgery, Minneapolis, 1908; aged 51; died, September 18, of coronary occlusion.

Arthur Thomas, Portland, Ore.; Physio-Medical College of Indiana, Indianapolis, 1895; veteran of the Spanish-American War; aged 72; died, September 14, of pneumonia.

Maurice Carlysle McKain ✠ Columbus, Ind.; Indiana University School of Medicine, Indianapolis, 1918; aged 48; was killed, September 25, in an automobile accident.

William Golliday O'Neal, Grenada, Miss.; Meharry Medical College of Walden University, Nashville, Tenn., 1903; aged 65; died, August 16, of carcinoma of the kidney.

Charles Edgar Keeler, Yakima, Wash.; University of Michigan Department of Medicine and Surgery, Ann Arbor, 1904; aged 68; died, August 10, of myocarditis.

Harry Leonard Baer ✠ Pittsburgh; University of Pittsburgh School of Medicine, 1917; served during the World War; aged 47; died, October 3, of coronary disease.

Gabriel Toombs Spearman, Mercedes, Texas; University of Georgia Medical Department, Augusta, 1900; aged 67; died, September 19, of sarcoma of the intestine.

Cora Millet Holden, Mentor, Ohio; Woman's Medical College of Pennsylvania, Philadelphia, 1882; aged 87; died, September 10, of carcinoma of the stomach.

Cesare Mondini, Brooklyn; Regia Università degli Studi di Palermo Facolta di Medicina e Chirurgia, Italy, 1888; aged 78; died, September 22, of cerebral hemorrhage.

Howard Davis Lewis, Sunbury, Pa.; University of Maryland School of Medicine, Baltimore, 1900; aged 62; died, October 2, of a self-inflicted bullet wound.

Samuel Edwin Walker, Somers Point, N. J.; Jefferson Medical College of Philadelphia, 1882; aged 86; died, September 6, of hypostatic pneumonia and senility.

R. Brent Murphy ✠ St. Louis; St. Louis Medical College, 1889; an Affiliate Fellow of the American Medical Association; aged 73; died, September 26, of carcinoma.

William F. Haley, Chicago; Chicago Homeopathic Medical College, 1889; aged 78; died, September 18, in Marion, Ohio, of arteriosclerosis and cerebral thrombosis.

Thomas Lewis Bowers, London, Ky.; Vanderbilt University School of Medicine, Nashville, Tenn., 1894; aged 74; died, October 16, of cerebral hemorrhage.

Leonidas F. Smith, Lindale, Ga.; Vanderbilt University School of Medicine, Nashville, Tenn., 1892; aged 72; died, September 20, in Rome of heart disease.

Victor A. Young ✠ Duluth, Minn.; Minneapolis College of Physicians and Surgeons, 1903; aged 65; died, September 20, of coronary sclerosis and hypertension.

Calvin Brobst Knerr, Philadelphia; Hahnemann Medical College of Philadelphia, 1869; aged 93; died, September 29, of bronchopneumonia and myocarditis.

Lester Barrett Klippel, Rochester, N. Y.; Syracuse University College of Medicine, 1919; aged 46; died, September 28, of coronary thrombosis.

William H. O'Malley, Kewanee, Ill.; College of Physicians and Surgeons of Chicago, 1893; aged 72; died in September of coronary thrombosis.

Collin Henly Wilcox, Daytona Beach, Fla.; Rush Medical College, Chicago, 1888; aged 76; died, September 8, of pneumonia and hemiplegia.

George Washington Olive, Berry, Ala.; Medical College of Alabama, Mobile, 1888; aged 78; died, September 23, of chronic myocarditis.

Arthur Louis Barton, Los Angeles; Colorado School of Medicine, Boulder, 1897; aged 67; died, October 1, in an automobile accident.

Allen D. Johnson, Atlanta, Ga.; Georgia College of Eclectic Medicine and Surgery, Atlanta, 1881; aged 86; died, September 20, of senility.

Alfred W. Z. Thompson, Yakima, Wash.; University of Oregon Medical School, Portland, 1913; aged 61; died, August 17, of uremia.

Ralph Clarke Williams, East Quogue, N. Y.; Trinity Medical College, Toronto, Ont., Canada, 1898; aged 64; died, September 3.

John Henry Tilden, Denver; Eclectic Medical Institute, Cincinnati, 1872; aged 89; died, September 1, of carcinoma of the bladder.

Harry Daniel Hopkins, Waterbury, Vt.; Baltimore Medical College, 1901; aged 62; died, October 2, of coronary thrombosis.

Rudolph J. Lange, Lansing, Mich; Saginaw (Mich.) Valley Medical College, 1903; aged 61; died, September 18, of myocarditis.

Wolf Maslon, Brooklyn; Eclectic Medical College of the City of New York, 1897; aged 73; died, September 10, of acute anemia.

Sherrill L. Mitchell ✠ Rolla, Mo. (licensed in Missouri in 1884); aged 78; died, September 30, of myasthenia gravis.

Bureau of Investigation

A PROMINENT IMPOSTOR

Koba-Kuba Shows Up as Matsuzaka

In THE JOURNAL for Aug. 1, 1931, page 339, the Bureau of Investigation published an article entitled "The Case of Koba, Alias Kuba." In this article it was pointed out that Tsuneyoshi Koba, sometimes referred to as L. (Lawrence) Tsuneyoshi Koba, was born in Japan in 1900 and came to the United States in 1914, graduated from the Seattle High School in 1919 and obtained a B.S. degree from Columbia University in 1924. He then enrolled in the freshman class at Johns Hopkins University School of Medicine for the 1924-1925 session but in June 1925 was dropped from that school for failures and poor standing. Subsequently he displayed forged credentials from Johns Hopkins University, including a forged diploma, and in 1929 imposed on three New York hospitals by means of these false credentials. In 1930 he attempted to present them to the California state authorities but, because of their activities, left the state hurriedly when faced with prosecution for filing fraudulent credentials from Johns Hopkins University. The California medical board found that he placed an order with a local lithographer for a replica of a Johns Hopkins medical diploma. The facts developed in this case were discussed by Dr. Charles B. Pinkham, Secretary of the California State Board of Medical Examiners, in an address on "Fraudulent Credentials" given before the 1930 Congress on Medical Education, Medical Licensure, and Hospitals.

Koba was next heard from in Minneapolis as Tsuneo Kuba, M.D. While in Minneapolis, he claimed that he had recently arrived from Japan to make a tour of various clinics and hospitals and to take a postgraduate course in surgery. He presented himself to the authorities of the University of Minnesota and claimed to be the son of a prominent surgeon in Tokyo (Dr. Kichiziro Kuba, who, he said, was a classmate of Dr. Dandy of Johns Hopkins) and a graduate of a medical school in Tokyo, of which he submitted evidence in the form of a diploma. He was granted the privileges of the surgical department as a voluntary fellow and was assigned to the urologic service. Dr. Guy S. Ford, dean of the Graduate School, found, however, that Koba could present no credentials, so the man was at no time registered as a graduate student.

Prior to his contacting the University of Minnesota, Koba had served for a short time one Boyd Williams, M.D., in a cancer-cure concern known as "Dr. Williams' Sanatorium." Later he related in Minneapolis that he was being persecuted by Williams and that a man named Tsuneyoshi Koba had been going around the country posing as a physician and that he had been mistaken for this individual because of the similarity in names. He claimed, further, that he had met this other individual at a ball game in Los Angeles and had bought all his medical books, thus explaining the names in his books. He also wrote to Johns Hopkins Medical School stating that, owing to an unfortunate similarity in names he was being confused with Koba, and asked that school to notify the American Medical Association that he (Kuba) was not the impostor Koba. He also sent the Bureau of Investigation details of this supposed mistake in identity. When asked for further information by the Bureau of Investigation he did not answer its letter.

In connection with subsequent investigation at the University of Minnesota, Dr. Wangensteen, chief of the Department of Surgery at the University, forced Koba to sit for a photograph. Copies of this photograph were sent to California and Washington, as well as to Johns Hopkins University, and replies came back confirming the identification of Tsuneo Kuba as Tsuneyoshi Koba. Finally (thanks to the persistent activity of Dr. Kano Ikeda of St. Paul) Koba, under the name Tsuneo Kuba, was arrested in Fargo, N. D., on a warrant charging him with entry into the country under false pretenses. At the time he was reported to be acting as a valet to a wealthy resident of Fargo. In order to avoid deportation he confessed that he was Tsuneyoshi Koba, who came to the United States in 1914. The Bureau of Investigation closed its article with the comment "Where and under what name and with what forged credentials will Koba next turn up?"

On March 26, 1932, under the heading "New York City Medical News," THE JOURNAL published an item stating that Lawrence Tsuneyoshi Koba, alias Tsuneo Kuba, pleaded guilty to attempted forgery in the third degree in the Court of General Sessions, New York County, Part 6, before Judge Morris Koenig, and was sentenced to the penitentiary on February 16 for an indeterminate period. Two years later (in 1934), under the heading "New York City Medical News," THE JOURNAL published an item stating that Koba had recently been discovered acting as a junior fellow in surgery at Presbyterian Hospital, Columbia University Medical Center, using the name Akira Matuzaki.

In 1935 the Rochester (N. Y.) *Democrat and Chronicle* called attention to the fact that Tsuneyoshi Koba was booked in Rochester police headquarters on the charge of vagrancy and

1924 1931 1935 1939

The 1924 picture of Koba was taken when he was a freshman at Johns Hopkins University Medical School. The 1931 picture of Koba was taken when he was at the University of Minnesota. The 1935 picture of Koba was taken from the Rochester Democrat and Chronicle of June 11, which reproduced excerpts of THE JOURNAL article (Aug. 1, 1931). The 1939 picture of Matsuzaka was taken when he was a junior at Johns Hopkins University Medical School. The first two pictures appeared in the former JOURNAL article on Koba and when shown to individuals who knew the man pictured as Matsuzaka above they stated that he was the same individual pictured as Koba, according to a Rochester physician.

that he had presented himself at Rochester with a letter of recommendation and sought a fellowship at a Rochester hospital to continue his research work, according to the police authorities. He claimed to have been a former Davis Cup player on the Japanese tennis team. One thing that made the authorities suspicious of Koba was the fact that he was not playing tennis of a caliber one would expect of a Davis Cup player. Unable to give him a fellowship, hospital authorities permitted him to remain as a visiting student. In connection with this case a Rochester physician wrote to the American Medical Association and obtained the information which the Bureau of Investigation had on Koba and excerpts from the report were included in the newspaper article referred to above.

In October 1940 another Rochester physician inquired of the Bureau of Investigation concerning a Japanese by the name of Masao Matsuzaka and stated that he suspected Matsuzaka was the impostor written up in THE JOURNAL some years ago. A check of the files indicated that he presumably referred to the article on Koba, especially because Koba, when at the Presbyterian Hospital in 1934, used the name Akira Matuzaki and because he was located in Rochester in 1935. A copy of THE JOURNAL article was forwarded to the Rochester physician and his attention was also called to the article which appeared in the *Democrat and Chronicle* for June 11, 1935. This physician subsequently wrote and stated the following:

"I wish to say that my patient has positively identified him [Masao Matsuzaka], from the pictures in the [Koba] article which you sent me, as the same individual who has worked for him as a butler for the past four or five years. As a butler, he was most satisfactory and thoroughly honest. It is interesting that this individual was left entirely alone, over periods of three to four months at a time, in the house and at no time was anything ever missing or lost. His prowess as a tennis player was a source of interest to friends of the family and he instructed the children in the playing of tennis and, at various times during the summer months at Chatham, Mass., was continually in demand by the highest social sets, as a tennis partner and opponent on the courts of the Chatham Beach Club. In the spring of 1939 he signified his intention to stop work to enter medical school at Johns Hopkins and to this end persuaded his employer to help him financially."

According to a telegram received by this Rochester physician and signed by Alan M. Chesney, M.D., Dean, Johns Hopkins Medical School, "Masao Matsuzaka, Japanese student, was admitted to third year class September 1939 and dropped from school June 1940 for failure in surgery, obstetrics and pediatrics."

According to the physician, Matsuzaka returned to Rochester in the summer of 1940 and told his former employer that because of excellence in his work he had been chosen as a special postgraduate student in surgery, to work under Graham in St. Louis, and again requested aid. Subsequently Washington University Medical School wrote to the employer for recommendation as to his personality and qualifications, and the employer asked the physician to contact Johns Hopkins. Washington University was then made aware of his record the preceding year at Johns Hopkins. Word has since been received that he has been refused admission to Washington University Medical School.

It does not seem to be entirely out of order for the Bureau to repeat its question of 1931: "WHERE AND UNDER WHAT NAME AND WITH WHAT FORGED CREDENTIALS WILL KOBA NEXT TURN UP?" and add "And how soon?"

MISBRANDED PRODUCTS

Abstracts of Notices of Judgment Issued by the Food and Drug Administration of the United States Department of Agriculture

[EDITORIAL NOTE.—These Notices of Judgment are issued under the Food, Drug and Cosmetic Act and in cases in which they refer to drugs and devices they are designated D. D. N. J., cosmetics C. N. J., and foods F. N. J. The abstracts that follow are given in the briefest possible form: (1) the name of the product; (2) the name of the manufacturer, shipper or consigner; (3) the composition; (4) the type of nostrum; (5) the reason for the charge of misbranding, and (6) the date of issuance of the Notice of Judgment—which is considerably later than the date of the seizure of the product and somewhat later than the conclusion of the case by the Food and Drug Administration.]

Black and White Ointment.—Plough Sales Corporation, Memphis, Tenn. Composition included about 8.05 per cent of red mercuric oxide, whereas the label falsely claimed 10 per cent of this substance was present. For itching, soreness and burning accompanying ringworm, psoriasis and eczema (of external origin).—[*D. D. N. J., F. D. C. 90; May 1940.*]

Booth's Camphorated Oil.—J. F. Booth, Harbor Springs, Mich. Composition: in one specimen not more than 12.6 per cent of camphor and in another not more than 9.3 per cent. Misbranded, since the Pharmacopeia provides that camphorated oil shall contain not less than 19 per cent of this substance. Misbranded also in the representations that it was efficacious as an anodyne in rheumatic affection of the joints and that it was useful in rheumatism, swellings of the breasts or joints or in colds on the chest.—[*D. D. N. J., F. D. C. 96; May 1940.*]

Booth's Carbolic Salve.—J. F. Booth, Harbor Springs, Mich. Composition included 2.9 per cent of carbolic acid. Product held to be adulterated because labeled to contain 5 per cent of this substance. Misbranded because falsely represented to be useful for ulcers, boils, hemorrhoids and some other conditions.—[*D. D. N. J., F. D. C. 96; May 1940.*]

Booth's La Grippe & Cold Tablets.—J. F. Booth, Harbor Springs, Mich. Composition: acetanilid (1 grain per tablet), a small amount of salol, a quinone compound and a bromide, with red pepper, chalk and starch. Falsely represented as the best remedy for la grippe and for stimulating the liver and secretions to perfect action.—[*D. D. N. J., F. D. C. 96; May 1940.*]

Booth's Liniment.—J. F. Booth, Harbor Springs, Mich. Composition: essentially volatile oils (including peppermint, mustard and wintergreen), alcohol (36.1 per cent by volume) and chloroform (10.8 per cent). Falsely represented as a remedy for rheumatism, gout, weak joints, sore lungs and some other things.—[*D. D. N. J., F. D. C. 96; May 1940.*]

Booth's Liver Pills.—J. F. Booth, Harbor Springs, Mich. Composition: plant drugs including red pepper, nux vomica and a laxative drug. Falsely represented as a remedy for headache, dizziness, torpid liver, dyspepsia and some other disorders.—[*D. D. N. J., F. D. C. 96; May 1940.*]

Booth's Mentholated Cough Drops.—J. F. Booth, Harbor Springs, Mich. Composition: sugar lozenges flavored with menthol. Falsely represented as giving immediate relief in coughs, colds, hoarseness and sore throat.—[*D. D. N. J., F. D. C. 96; May 1940.*]

Cotec.—Cotec Co., Lynn, Mass. Composition: essentially fat and excrement. Falsely represented as a remedy for hemorrhoids, sores, ulcers, prolapsis of the bowels, kidney and bladder trouble and some other things.—[*D. D. N. J., F. D. C. 95; May 1940.*]

Dormalgin.—Lawson M. Luth, Geneva, N. Y. Composition: aminopyrine and a barbituric derivative. Falsely represented as a useful, harmless pain killer, whereas it contained dangerous drugs.—[*D. D. N. J., F. D. C. 97; May 1940.*]

Ma-El-Ra-Tone Herb Compound.—General Products Laboratories, Columbus, Ohio. Composition: a material proportion of epsom salt, a mineral substance, whereas the label reported it to consist entirely of herb and other vegetable substances.—[*D. D. N. J., F. D. C. 93; May 1940.*]

Messina Effervescente Granulare.—Drew Corporation, Brooklyn. Composition: According to the label, it was prepared with sugar, baking soda, tartaric acid, citric acid and oil of lemon. The undeclared presence of an additional substance, borax, was found to constitute misbranding.—[*D. D. N. J., F. D. C. 92; May 1940.*]

Myasthene Tablets.—Medicinal Specialties Co., New York. Composition: 7.2 grains of aminoacetic acid (glycocoll) per tablet. Falsely represented as efficacious for "tired feeling," loss of appetite and certain types of nervousness.—[*D. D. N. J., F. D. C. 100; May 1940.*]

Old Man Frantz Mountain Tonic.—Old Man Frantz, Pittsburgh. Composition: in each fluid ounce 178 U. S. P. units of vitamin A, 400 international units of vitamin B_1, 334 international units of vitamin C, and not more than 251 U. S. P. units of vitamin D. Falsely represented to contain a significant amount of vitamin A per prescribed dose; to increase vigor and remedy such conditions as nervousness, poor appetite, dry skin, diarrhea, sterility, glandular atrophy, pellagra, dermatitis, loss of hair, and many other things.—[*D. D. N. J., F. D. C. 85; May 1940.*]

Saurinol.—Saurinol Distributors Corporation (no address given). Composition: "consisted essentially of medium boiling petroleum oil." Falsely represented as a relief from sinus trouble, hay fever, exposed cancer, varicose veins, pyorrhea, trench mouth, laceration, ulcers, and skin diseases.—[*D. D. N. J., F. D. C. 98; May 1940.*]

Seeley's Spook Oil Linament.—G. A. Seeley, Louisville, Colo. Composition: turpentine (50 per cent) wintergreen (2 per cent) and a fatty oil. Falsely represented as useful for treating hemorrhoids, toothache, colds, sunburn, earache, sore joints, rheumatic pains, dandruff and some other things.—[*D. D. N. J., F. D. C. 101; May 1940.*]

Yucca-Balm.—Geo. Bell Co. (Yucca Balm Co.), Ogden, Utah. Composition: essentially soft soap and cresol (0.30 per cent). Falsely represented as a relief of sore feet, dandruff, pains and swellings.—[*D. D. N. J., F. D. C. 102; May 1940.*]

Queries and Minor Notes

THE ANSWERS HERE PUBLISHED HAVE BEEN PREPARED BY COMPETENT AUTHORITIES. THEY DO NOT, HOWEVER, REPRESENT THE OPINIONS OF ANY OFFICIAL BODIES UNLESS SPECIFICALLY STATED IN THE REPLY. ANONYMOUS COMMUNICATIONS AND QUERIES ON POSTAL CARDS WILL NOT BE NOTICED. EVERY LETTER MUST CONTAIN THE WRITER'S NAME AND ADDRESS, BUT THESE WILL BE OMITTED ON REQUEST.

PLANO LENS IN SAFETY SPECTACLES

***To the Editor:*—With regard to safety spectacles used in industries, with a six-curve plano lens is it true that it will produce a prismatic effect, any magnification at reading distance and surface reflections regardless of the frame and its size? I have an example of three patients who have a vision of 20/20 or better in each eye who complain of a pulling sensation while wearing the plano goggles. None have any disease.**

M.D., New York.

ANSWER.—This question is really a series of several questions, answers to which will be taken in order:

1. True plano lenses cannot produce prismatic effects. A "plano" lens ground on a six base curve has usually a slight myopic effect. Several manufacturers, when questioned, agreed that the presence of an additional slightly smaller image sometimes seen through such lenses is due to the light bouncing back and forth between the two surfaces of the lenses and finally emerging so as to produce the apparent effect of a prism. The secondary images seen at the side are always much smaller and much diminished in intensity. They may be somewhat further diminished by the use of a thin layer of soap or by the use of a tint in the glass. In plus lenses (lenses with a convex effect) one practically never notices this phenomenon.

2. True plano lenses produce no magnification at the reading distance or at any other distance.

3. Surface reflections are not due to the size of the lens or to the frame in which it rests.

4. The pulling sensation complained about by patients wearing true plano goggles may be due to one of two causes: Either the goggles are not perfectly ground, that is they contain flaws such as waves or pits or slight irregularities, or there are actual prisms in the goggles. If the goggles are not perfectly ground, flaws or a slight minus effect make them exceedingly annoying. This is often the case with cheap goggles. When there are prisms in the goggles, the lenses are not plano but have an actual prism. Usually this prism has a base in the vertical position, as a small prism base in or out almost never causes any discomfort. If it does, it is usually considered to be entirely on a psychologic basis.

Large companies who fit many goggles find that men between 18 and 30 are apt to complain of a pulling sensation. In these plants it is customary to have the correction ground into the goggles. Even then occasionally there are complaints and this is particularly true if the goggles are incorrectly ground or poorly surfaced.

ALLERGIC RHINITIS FROM READING NEWSPAPER

***To the Editor:*—A patient with allergic rhinitis gets sneezing spells from reading a freshly printed newspaper or a magazine with heavy print. The patient is able to read the newspaper without trouble a day after it has been opened and exposed to the air. Which part of the print could be the offending substance and what tests would you recommend?**

M.D., Wisconsin.

ANSWER.—The printing ink may be the offending cause of the rhinitis, although customarily dermatitis rather than rhinitis results from such exposure. Colored and rotogravure inks are more prone than black inks to induce manifestations, but in this instance the implication in the query is that the exposure is to black ink, since reference is made to newspapers. The composition of black ink varies appreciably, but typical ink consists of carbon black (at times coal tar) with some resin such as shellac in an oil such as linseed with a quick drier such as turpentine. It is suggested for test purposes that a small quantity of black ink be procured from some pressroom and be smeared on a glass surface. This should be located in the patient's room in order to establish any response. If the characteristic rhinitis promptly appears, this may be sufficient for diagnostic purposes, but otherwise similar tests may be carried out with respective ingredients, such as shellac, turpentine or lamp black. If no response follows exposure to the ink, a few sheets of blank newsprint paper similarly should be acquired from the pressroom, which should be handled by the patient as though it were a newspaper. In pressrooms, printers occasionally develop respiratory disorders from paper pulp dust, which, however, obviously is more prevalent in a pressroom than from handling a single newspaper. Should these tests fail, it should be recognized that printing presses are being cleaned continually with such solvents as naphtha, benzene, toluene, soda ash and trisodium phosphate. It is unlikely that these cleaning agents would carry over to the printed page, but the possibility exists. If all such tests disclose no cause for the rhinitis, it may be helpful to carry out tests with a great variety of ingredients of colored inks, which vary from color to color. A list of the commoner constituents of colored ink may be found in Schwartz and Tulipan's "Occupational Diseases of the Skin." Among others, there are mentioned paranitranilin, Persian berries, Brazilian wood, redwood and picric acid.

Some magazines but, as far as known, no newspapers, are printed by the offset method. In this process the printed sheet may be sprayed with a suspension of acacia or starch in alcohol. Sometimes printers develop respiratory disorders, chiefly asthma, following sensitization to these vegetable dusts. Conceivably, enough of such drying agents might adhere to the page to harm the patient.

LIBIDO IN ELDERLY WOMAN

***To the Editor:*—Could you offer some suggestion as to treatment and possible etiology of a persistent intense libido in a widow aged 72? Examination does not show any genital irritation which might be an exciting factor. Symptoms started following administration of "Cortalex" given for daily fainting spells which were preceded by a feeling of pronounced weakness. Treatment consisted of one tablet of Cortalex four times daily for three weeks. Results were good. About one month later the patient started to complain of a tingling sensation and itching of the breast and later the increased libido. The breasts seemed to enlarge temporarily; also the patient complained of an enlargement of the abdomen, first noticed when her clothes became tight. This has since decreased by 2 inches actual measurement. The breasts have since returned to normal size, although at times the tingling returns to such a degree that the patient slaps her breasts for relief. The increased libido either follows or accompanies this occurrence. There is a history of salpingectomy following an old gonorrheal infection; also a second operation for adhesions. Pelvic examiation shows normal hair distribution, normal external genitalia and normal vaginal mucosa for a person of this age; the vaginal introitus and canal are tight. The uterus is apparently atrophied but smooth in outline and in normal position. There is no cystocele or rectocele and no sign of pelvic tumor or any pronounced abdominal tenderness. Spinal fluid pressure is normal. Blood and spinal fluid Wassermann tests are negative. Hemoglobin and blood counts are normal. The blood pressure is 112 systolic, 70 diastolic. Otherwise the physical examination is essentially negative. The patient is nervous; she has had two illnesses which she described as nervous breakdown and has little to do to occupy her time. Her appetitie is poor. Bromides and barbitals together with the use of a soothing vaginal suppository are all ineffective. Testosterone propionate is the only thing that seems to give some relief. Is there any contraindication? Do you have any other suggestions?**

M.D., Washington.

ANSWER.—It is highly doubtful whether the increase in libido following the administration of adrenal cortex extract can be explained on the basis of a stimulation in growth of the reproductive organs. Adrenal cortex steroids have a slight proliferating effect on the sex organs but it is improbable that the amount of activity in the dosage used by this patient would have any significant effect in this direction. It is difficult to explain the result obtained. One possibility is that the extract produced water retention in the various tissues of the body, including the vagina and clitoris. This water retention may not be visible grossly. Nevertheless it could cause an irritation which would result in the increased libido. Ammonium chloride 0.5 Gm. (7½ grains) three times a day might be administered, as recommended by Greenhill and Freed for the relief of premenstrual edema and tension (*Endocrinology* **26**:529 [March] 1940) in the treatment of this patient. If this therapy is effective the theoretical consideration that the libido was due to the increase in extracellular fluid about the vulva would appear justified.

SUBCUTANEOUS ATROPHY FROM INSULIN

***To the Editor:*—Certain individuals are susceptible to subcutaneous atrophy from the use of insulin. I am anxious to find out what this X product is that causes this subcutaneous atrophy. I believe it could be used in certain individuals, especially from a cosmetic standpoint, and I would appreciate it if you would give me the names of some laboratories which you think would be interested in research along this line.**

William VanderVoort, M.D., Battle Creek, Mich.

ANSWER.—This inquiry may be best answered by quoting from the recently published book by Wilder, Clinical Diabetes Mellitus and Hyperinsulinism. Under the heading "Insulin Fat Atrophy" on page 95 the following statement appears: "By insistence on varying the site of injection, the occurrence of these disfiguring lesions usually can be avoided. However, they have been encountered, even with well trained patients. The cause is unknown. Some type of local sensitivity of the tissues of an allergic type may be responsible. Almost all these patients have been women. The treatment of insulin atrophy is principally a matter of prevention by the exercise of scrupulous care in the

technic of the hypodermic infection. There is some advantage in using only highly concentrated insulin, such as U-80. If the affected regions are avoided in subsequent treatment, the depressions after several months usually fill in spontaneously; occasionally they persist for years."

The suggestion that an X product in insulin is responsible for this subcutaneous atrophy is not a likely one. The condition occurs in so few individuals that the abnormality is probably in the patient rather than in the product. Even if there were such an X product it would be most unwise to use it as a cosmetic agent for the reason, as Joslin has emphasized, that occasionally the atrophic regions are remote by several centimeters from the site of injection.

LEUKOPENIA AND RELATIVE LYMPHOCYTOSIS

To the Editor:—A girl aged 5 years, weighing 42 pounds (19 Kg.), had severe scarlet fever Feb. 1, 1940. The white blood count was 42,000, polymorphonuclears from 78 to 80 per cent, eosinophils from 10 to 12 per cent. On the fourth day of illness the child was put on sulfanilamide 45 grains (3 Gm.) daily for three days, then 30 grains (2 Gm.) daily for three days, then 20 grains (1.3 Gm.) daily for two days and 15 grains (1 Gm.) for one day. White blood counts were done daily during the administration of sulfanilamide. At the end of the treatment with sulfanilamide the white blood count was 22,000, polymorphonuclears from 79 to 80 per cent. The blood picture was not unusual and the patient was doing well. February 21 the temperature suddenly went to 104. The patient was severely nauseated and vomited considerably for one day. She had a moderate cervical adenitis. The white blood count was 36,000. She was again put on sulfanilamide 30 grains (2 Gm.) for three days. White blood counts and differentials were done daily. At the end of four days the patient was again doing well and the white blood count was 21,000. There was no reduction in the polymorphonuclears. The patient had no fever; was up and about and felt well. June 13 and June 23 she was given typhoid vaccine. June 28, 29 and 30 she had a fever of 102. She did not feel particularly bad and had no physical signs. July 1 the patient's temperature was normal and she felt well. On this date the red blood count was 4,100,000, hemoglobin 70 per cent, white blood count 6,100, polymorphonuclears 22.5 per cent, lymphocytes 64 per cent, monocytes 7 per cent, eosinophils 6.5 per cent, basophils none. July 3 the white blood count was 8,450, polymorphonuclears 25 per cent, lymphocytes 55 per cent, monocytes 11.5 per cent, eosinophils 8.5 per cent. Is it possible that the leukopenia, neutropenia and increase in the number of lymphocytes could be caused by the typhoid vaccine? It hardly seems possible that the neutropenia could be caused from the sulfanilamide; however, no count was done one month after the sulfanilamide was discontinued. The patient is feeling well and has no sore throat. She is on a general supportative treatment. What is the significance of the reduced neutrophils?

John E. Haynes, M.D., Dawson Springs, Ky.

ANSWER.—Several explanations of the leukopenia and relative lymphocytosis in this patient are possible:

1. She may have had some systemic infection such as influenza, which is characterized by such a blood picture. Certainly her temperature beginning five days after a typhoid vaccine injection is an unusual reaction, and the vaccine cannot be assumed to be the cause of this.

2. Following a severe infection, a lymphocytosis is frequently seen and may last for several months.

3. Lymphocytosis is usual in children. Certainly it is unlikely that the sulfanilamide played a part. The typhoid vaccine may have been partially responsible.

TUBERCULOUS MENINGITIS

To the Editor:—Are there authentic records of death from tuberculous meningitis with an absence of clinical symptoms or records of tuberculous meningitis which show profuse hemorrhage into the spinal canal together with definitely formed blood clots? M.D., New Jersey.

ANSWER.—If the meaning of the inquiry is that patients die of meningitis with no symptoms at all, the answer is no; that is, there is no record of a fatal symptomless tuberculous meningitis. There are rare reports, however, on a healing meningitis, but not without symptoms (Hektoen, Ludvig: The Fate of Giant Cells in Healing Tuberculous Tissue, *Tr. Chicago Path. Soc.* **2**:283, 1897). If it is meant that there are no other symptoms than those originating within the cranium, then the answer is yes. Many cases develop from old tuberculomas beneath the ependyma long after the other body foci are healed (Rich, A. R., and McCordock, H. A.: The Pathogenesis of Tuberculous Meningitis, *Bull. Johns Hopkins Hosp.* **52**:5 [Jan.] 1933).

It is also possible for tuberculous meningitis to be found at necropsy in the absence of clinical symptoms. This rare occurrence represents an early stage incidental to an advanced pulmonary tuberculosis wherein symptoms of the latter were so severe that they overshadowed those of the meningitis. As most of the symptoms of meningitis are due to intracranial pressure, it is thus possible for the early lesions to exist without significant symptoms.

No reports of profuse hemorrhage into the spinal canal in tuberculous meningitis are available and they have not been seen in extensive experience in large tuberculosis sanatoriums with or without blood clots. It is possible, however, that they may result during a meningitis or from intracranial trauma, convulsions, falling against or out of bed, hemorrhagic diathesis or cerebral hemorrhage.

NEUROLOGIC EFFECTS OF PELLAGRA

To the Editor:—What is the cause of the combined degeneration of the spinal cord in pellagra? What is the cause of the macrocytic anemia which occasionally appears in pellagra? Of the cerebral symptoms in pellagra, which are caused by nicotinic acid deficiency and which are caused by thiamine hydrochloride deficiency? Is "central neuritis" generally accepted terminology? M.D., California.

ANSWER.—The cause of the combined degeneration of the spinal cord in pellagra is not known. Sufficient time has not elapsed since the discovery of the crystalline vitamins to ascertain whether any one of the known factors in the vitamin B complex is responsible or not. The macrocytic anemia which occasionally appears in pellagra may arise from a deficiency of intrinsic factor such as in addisonian conditions of anemia or may be due to a lack of the factor described by Wills. Of the cerebral symptoms in pellagra it is not yet known which ones are caused by lack of nicotinic acid and which by lack of thiamine hydrochloride, but there seems to be a degree of nonspecificity in that similar conditions may be relieved by either one of these compounds. The term "central neuritis" is well known to workers in the fields of psychiatry and neurology.

References:

Wills, Lucy; Clutterbuck, P. W., and Evans, Barbara D. F.: A New Factor in the Production and Cure of Macrocytic Anemias and Its Relation to Other Hemopoietic Principles Curative in Pernicious Anaemia, *Biochem. J.* **31**: 2136 (Nov.) 1937.

Frostig, J. P., and Spies, T. D.: The Initial Nervous Syndrome of Pellagra and Associated Deficiency Diseases, *Am. J. M. Sc.* **199**: 268 (Feb.) 1940.

CONGENITAL SYPHILIS IN ADULT WOMAN

To the Editor:—A 28 year old woman at present is in good health and has no complaint with the exception that glasses must be worn all the time. Fourteen years ago on March 17, 1926, a 4 plus Wassermann reaction was discovered during the investigation of a severe iritis and some arthritis. Her mother's blood also was found to be 4 plus. Treatment was instituted at once and consisted in the injection of eighteen doses of neoarsphenamine of 0.45 Gm. each during the course of the next seven months. There is no certain knowledge of the serologic reaction at this time although the patient is sure it was negative. Two years later, in March 1928, the Wassermann reaction of the blood was recorded as 3 plus. At this time the patient was started on mercuric iodide and mercury rubs. The number of applications is not known but the patient states that treatment continued for many weeks and that at the end the blood reaction was negative. The only residual effect was a disturbance in vision for which glasses have had to be worn since. The iritis cleared rapidly under the neoarsphenamine. In the past few years the patient has had several Kahn and Wassermann tests at reliable laboratories and all have been negative. Two spinal fluid Wassermann tests have been done in the past two years and were negative. Complete physical examinations have been done in the past three or four years and the only defect noted is in the eyes. The pupils are slightly irregular and are small. They do not react to light or in accommodation. (Am I correct in the belief that this is not an Argyll Robertson pupil since it does not react in accommodation?) The eyegrounds appear to be quite normal. The patellar and other reflexes are normal. Blood counts and urine examinations give normal results. The remainder of the physical examination is normal. X-ray examinations of the chest and heart about a year ago gave normal results. What should be the program for this patient from now on?

Ralph E. White, M.D., Santa Ana, Calif.

ANSWER.—The question of subsequent treatment of a 28 year old woman with congenital syphilis in whom the blood and spinal fluid tests are negative and who presents no active symptoms of the disease depends in a great measure on whether or not she is married and is likely to become pregnant or is desirous of becoming pregnant.

If unmarried, there are two possible courses to pursue: one, to give fifteen injections of a bismuth compound twice a year for three years and then observe regularly thereafter; the second course and the one that seems warranted for this young woman is to give no treatment but to continue only with observation, having her report every six months for a recheck. The status of the blood serologic tests whether negative or positive are not of significance under this program.

On the other hand, if she is married or plans to marry, and if she becomes pregnant intensive treatment should be given. The treatment is given not only to assure her of a nonsyphilitic child (for which the prospects are excellent) but, of equal importance, to prevent a recurrence of the trouble in her eyes, whether it is iritis or interstitial keratitis. The assumption that she does not have Argyll Robertson pupils seems valid, as the absence of pupillary reactions might well be the result of the iritis she has had.

RELAPSE OF DIABETES

To the Editor:—**A youth aged 18 came under my care in February 1940 with severe diabetes mellitus. His original fasting blood sugar was 362 mg. during institutional treatment. He quickly responded to ordinary therapy and was discharged on a liberal diet varying from 2,300 to 2,800 calories and 30 units of protamine insulin daily, under which his blood sugar was normal and there was no glycosuria. The patient is an athlete and his cooperation in all respects has been excellent owing to his desire to reattain physical efficiency. Over a period of several months his course was entirely uneventful until he became employed in a pea canning factory. He was here engaged in making syrup, the process consisting of the addition of refined granulated sugar and salt to boiling water. He worked in a steam enclosed room. Forty-eight hours after undertaking this work his urinary sugar sharply increased and all symptoms of severe diabetes mellitus made their appearance. I am quite positive that there was no intentional intake of sugar and that there could have been no source of sugar consumption other than by the inhalation of a sugar-saturated atmosphere. Careful cleansing of the hands had been done previous to eating. Would it be possible by the means of such sugar-saturated steam inhalation to bring about this marked glycosuria?**

M.D., Illinois.

ANSWER.—A diabetic patient who changes his mode of life is apt to suffer a temporary upset of the control of his diabetes. The amount of exercise, hours for meals and actual content of meals may vary and it takes time to become adjusted.

Sugar is not volatile and would not be present in the steam from the boiling liquid. One could not explain on that basis the flare-up in the diabetic condition. Theoretically the patient should not have an increase in glycosuria simply because of working in the atmosphere described.

Still other factors enter into the problem. Any diabetic person energetically treated at the onset of his diabetes is apt to do surprisingly well. After a few months, adjustments take place and the results are not quite as clearcut, owing to the natural course of the disease, to gradual laxity in treatment by the patient, or to the fact that the tolerance for carbohydrate has been overestimated and the prescribed diet increased above the optimum for ease of control.

PHOTOPHOBIA

To the Editor:—**For more than five years I have suffered from photophobia. This appeared to have started while using a microscope in medical school about 1928. Glasses for hyperopia were prescribed. Little or no relief was obtained. At present I am wearing a + 1.50 in both eyes, no tinting, with no relief (latent hyperopia—vision without cycloplegic is 20/15). The condition is much worse after a period of reading or picture shows; that is, after use of the eyes. On waking there is extreme sensitivity to light for about a half hour, after which I am able to open the eyes fairly well. Glasses have been prescribed by several competent ophthalmologists, usually with disagreement as to the amount of correction, type of frames, tinting and the like. My general health is good. There is strong possibility of a chronic (if there is such) appendicitis and possibly a focus of infection in the prostate. I must study at least an hour a day.**

M.D., Georgia.

ANSWER.—Photophobia in a large percentage of cases is due to a chronic inflammatory process of the conjunctiva, with or without minute infiltrations of the superficial cornea. These are apt to be overlooked in the course of a routine refraction; in fact in many cases such forms of keratitis can be detected only with a slit lamp. Again, in certain instances photophobia may result from the train of conditions attendant on an imbalance of the ocular muscles. Proper attention to the muscle balance and fusion will be followed by permanent relief. In only a few cases is photophobia the result of a true retinal sensitivity. This may be due in some instances to glandular dysfunction, to vitamin deficiency or occasionally to a chronic focal infection. Consequently the search for the cause of photophobia should be directed to the conjunctiva, the cornea, the muscle function and, finally, if all of these are found to be normal, to a possible source of retinal irritation.

TESTING FOR ARCH STRENGTH OF FEET

To the Editor:—**In connection with the examination of army applicants it is often difficult to determine the strength of the arches in cases of congenital or acquired flatfoot. What tests are employed by orthopedists to determine the strength or weakness of such feet?**

M.D., Washington.

ANSWER.—There has been no satisfactory test devised to determine the strength or weakness of feet of army applicants. The only satisfactory test is that of the functional capacity. For the present this is best judged from the history, that is, how far the individual can walk. The appearance of the foot is to some degree an indication of the capacity of the foot. A weak foot of long existence will show pes valgoplanus. On the other hand, a weak foot that has had no great demand made on it could show a relatively good appearance, particularly if it has been supported. The pes valgoplanus in every case is a less efficient foot than one that retains the normal position with weight bearing. An indication of the amount of decompensation of the foot can be obtained by measuring the foot before and after weight bearing, both as to length and as to width. Another method of measurement is to take lateral roentgenograms of the foot with the foot on a flat surface, without weight bearing and then with weight bearing, and determine the height of the arch in each case.

Unfortunately, these methods have not been developed to an extent to which they can be used as a direct indication of the capacity of the foot. At the present time there is no objective method of determining the actual strength of a flatfoot except by its ability to carry out the work required.

PTERYGIUM

To the Editor:—**One of my patients states that he had bilateral pterygium cured within two weeks by using topical applications prescribed by a specialist and that he did not undergo any operative procedure whatever. Are there any drugs which may be used successfully in the treatment of pterygia? If so what are they and are they safe to use—or, rather, what are their limitations?**

Stanley H. Macht, M.D., Crewe, Va.

ANSWER.—Cases of true pterygium are not cured by any topical application so far as known. They have been treated by electrocoagulation, which obliterates the vessels and causes the tissue to slough off, but this method has nothing to recommend it over a properly performed surgical procedure. It is contended that the use of some strong coagulant, such as trichloroacetic acid, might also obliterate the vessels, but it is not considered as safe as a proper surgical procedure.

REPORTING RESULTS OF TESTS FOR SYPHILIS

To the Editor:—**The Committee on the Evaluation of Serodiagnostic Tests for Syphilis of the United States Public Health Service has advocated reporting the results of all serum tests for syphilis as "positive," "doubtful" and "negative." Under this plan all definitely positive reactions hitherto recorded as strongly or moderately positive are reported merely as "positive," while weaker reactions are reported as "doubtful." I would appreciate greatly an interpretation of "strongly" and "weaker" reactions as compared with the usual 4 plus, 3 plus, 2 plus, 1 plus, plus-minus. In other words, how should a 3 plus reaction, how a 2 plus reaction be reported according to the plan of the committee?**

C. Alexander Hellwig, M.D., Wichita, Kan.

ANSWER.—Serologists have not adopted a single plan for reporting the results of tests for syphilis. Kolmer, for example, reports very strongly positive, strongly positive, moderately positive, weakly positive, doubtfully positive and negative. Kahn reports positive (++++), positive (+++), positive (++), doubtful (+), doubtful (±) and negative. Other serologists, as for example in the New York State Health Department Laboratories, report quantitative serologic titers. However, those who report positive, doubtful and negative generally include 4 plus, 3 plus and 2 plus reactions under positive, 1 plus and plus-minus reactions under doubtful.

ISOTHIOCYANATES IN COLD CREAM

To the Editor:—**Would the incorporation of an isothiocyanate in a cold cream base be injurious to the skin? Any information and references would be appreciated.**

Jacob Weinless, M.D., Bronx, N. Y.

ANSWER.—It has been shown that the rubefacient action of dissolved allylisothiocyanate is roughly inverse to its solubility in a given solvent. Thus, a 1 per cent solution of allylisothiocyanate in olive oil causes little or no irritation; in dilute alcohol marked irritation; in an emulsion in mucilage, still more irritation. It therefore might be practical to incorporate esters of isothiocyanic acid with cold creams in order to obtain controlled rubefacient action such as would be desirable in certain chronic cutaneous conditions in which increased circulation in the blood vessels of the skin is desired. The esters (allylisothiocyanate) are commonly called the volatile oil of mustard and are strong irritants and vesicants. The use of isothiocyanates in cold creams is not to be recommended as a general practice.

ASTHMA IN PREGNANCY

To the Editor:—**A primipara aged 35 is a sufferer from asthma. Her blood pressure is 120 systolic, 70 diastolic. What effect if any will epinephrine have on the uterus and what effect, if any, will solution of posterior pituitary have on the asthma? What anesthetic would be suitable for this case, for home delivery?**

M.D., Kentucky.

ANSWER.—Solution of epinephrine in therapeutic doses is not contraindicated during pregnancy because of any possible abortion. The use of solution of posterior pituitary in asthma is not considered of benefit by most workers in the field. The choice of an anesthetic is not affected by asthma in the absence of respiratory infection. General ether anesthesia is recommended by some allergists for temporary relief of severe asthma.

Medical Examinations and Licensure

COMING EXAMINATIONS

BOARDS OF MEDICAL EXAMINERS
BOARDS OF EXAMINERS IN THE BASIC SCIENCES

Examination of boards of medical examiners and boards of examiners in the basic sciences were published in THE JOURNAL, November 9, page 1658.

NATIONAL BOARD OF MEDICAL EXAMINERS

NATIONAL BOARD OF MEDICAL EXAMINERS: Parts I and II. Various centers, February. Part III. Boston during November. Exec. Sec., Mr. Everett S. Elwood, 225 S. 15th St., Philadelphia.

EXAMINING BOARDS IN SPECIALTIES

AMERICAN BOARD OF ANESTHESIOLOGY: *Written.* Various centers, Feb. 20. Final date for filing application is December 21. *Oral.* Part II. Cleveland, May 31-June 1. Final date for filing application is April 1. Sec., Dr. Paul M. Wood, 745 Fifth Ave., New York.

AMERICAN BOARD OF DERMATOLOGY AND SYPHILOLOGY: *Oral.* Chicago, Dec. 6-7. Applications for Group A are closed. Sec., Dr. C. Guy Lane, 416 Marlboro St., Boston.

AMERICAN BOARD OF INTERNAL MEDICINE: *Written.* Parts 1-A and 1-B, Feb. 17. Final date for filing application is Jan. 1. Sec., Dr. William S. Middleton, 1301 University Ave., Madison, Wis.

AMERICAN BOARD OF OBSTETRICS AND GYNECOLOGY: Part II, Groups A and B, Cleveland, May 28-June 1. Final date for filing application is March 15. Sec., Dr. Paul Titus, 1015 Highland Bldg., Pittsburgh.

AMERICAN BOARD OF OPHTHALMOLOGY: *Oral.* Cleveland, May or June. *Written.* Various centers, March 8. *The only written examination during 1941.* Applications must be on file not later than Dec. 1. Sec., Dr. John Green, 6830 Waterman Ave., St. Louis.

AMERICAN BOARD OF PATHOLOGY: *Oral and Written.* Cleveland, June 1-2. Final date for filing application is May 1. Sec., Dr. F. W. Hartman, Henry Ford Hospital, Detroit.

AMERICAN BOARD OF PEDIATRICS: New York, March 30-31, following the Region I meeting of the American Academy of Pediatrics. Chicago, May 18, following the Region III meeting of the American Academy of Pediatrics. Sec., Dr. C. A. Aldrich, 723 Elm St., Winnetka, Ill.

Illinois June Report

Mr. Lucien A. File, superintendent of registration, Department of Registration and Education, reports the written examination for medical licensure (graduates of foreign schools given also a practical test) held in Chicago, June 25-27, 1940. The examination covered ten subjects and included 100 questions. An average of 75 per cent was required to pass. Two hundred and ninety-nine candidates were examined, 292 of whom passed and seven failed. The following schools were represented:

School	PASSED	Year Grad.	Per Cent
College of Medical Evangelists		(1940)	83
Chicago Medical School		(1940)	78, 78, 78, 78, 79, 80, 80, 80, 81, 81, 81, 81, 81, 81, 82, 82, 82, 83,* 83, 83, 83, 83, 83, 83, 83, 83, 84,* 84, 84, 84, 84, 84, 84, 84, 85,* 85
Loyola University School of Medicine		(1939) (1940)	83, 76,* 77, 77, 77, 78, 79, 79, 79, 79, 80, 80, 80, 80, 80, 80, 80, 80, 81,* 81, 81, 81, 81, 81, 81, 81, 81, 81, 81, 81, 82, 82, 82, 82, 82, 82, 82, 82, 82, 82, 83, 83, 83, 83, 83, 83, 83, 84,* 84, 84, 84, 84, 84, 84, 84, 85, 85, 85, 88
Northwestern University Medical School		(1939) (1940)	82,* 83,* 84, 84, 80, 81, 81, 82, 82, 82, 83,* 83, 83, 83, 83, 83, 84,* 84, 84, 84, 84, 84, 84, 85, 85, 85, 86,* 86, 86, 87
Rush Medical College		(1938) (1939)	85, 87, 79, 80, 81, 81, 81, 82, 82, 82, 83, 84, 85, 85, 85, 85, 85, 85, 85, 86, 86
University of Chicago, The School of Medicine		(1939)	81, 85,* 86*
University of Illinois College of Medicine		(1939) (1940)	83, 84, 77, 78, 78, 79, 79, 80,* 80, 80, 80, 81, 81, 81, 81, 81, 81, 81, 81, 82,* 82,* 82,* 82,* 82, 82, 82, 82, 82, 82, 82, 82, 82, 82, 82, 82, 82, 82, 83,* 83,* 83, 83, 83, 83, 83, 83, 83, 83, 83, 83, 83, 83, 83, 83, 83, 83, 83, 83, 83, 84,* 84, 84, 84, 84, 84, 84, 84, 84, 84, 84, 84, 84, 84, 84, 85,* 85,* 85, 85, 85, 85, 85, 85, 85, 85, 85, 85, 85, 85, 85, 85, 86, 86, 86, 86, 86, 87, 88
Indiana University School of Medicine		(1938)	83
University of Oklahoma School of Medicine		(1939)	79*
Hahnemann Med. College and Hospital of Philadelphia		(1939)	78, 78
University of Pennsylvania School of Medicine		(1938)	84
Marquette University School of Medicine		(1940)	80
University of Wisconsin Medical School		(1939)	80
University of Toronto Faculty of Medicine		(1939)	84
Karl-Franzens-Universität Medizinische Fakultät, Graz		(1929)	77
Medizinische Fakultät der Universität Wien		(1911) (1915) (1921) (1924) (1925) (1926) (1932) (1933) (1937) (1938)	79, 80, 79, 80, 76, 76, 81, 81, 82, 79, 84, 80, 81
Deutsche Universität Medizinische Fakultät, Prag		(1937)	76
Albert-Ludwigs-Universität Medizinische Fakultät, Freiburg		(1917)	75
Friedrich-Wilhelms-Universität Medizinische Fakultät, Berlin		(1920) (1937)	79, 79
Julius-Maximilians-Universität Medizinische Fakultät, Würzburg		(1922)	80
Johann Wolfgang Goethe-Universität Medizinische Fakultät, Frankfurt-am-Main		(1926)	81
Kaiser-Wilhelms Universität Medizinische Fakultät, Strassburg		(1902) (1917)	76, 78
Schlesische-Friedrich-Wilhelms Universität Medizinische Fakultät, Breslau		(1917) (1926)	79, 75
Universität Heidelberg Medizinische Fakultät		(1932) (1938)	77, 77
Vereinigten Friedrichs-Universität Medizinische Fakultät, Halle-Wittenberg		(1924)	80
Magyar Királyi Erzsébet Tudományegyetem Orvostudo-Mányi, Pecs		(1926)	77
Regia Università di Pisa Facoltà di Medicina e Chirurgia		(1935)	77
Licentiate of the Royal College of Physicians, of the Royal College of Surgeons of Edinburgh and of the Royal Faculty of Physicians and Surgeons of Glasgow		(1938)	81
Universität Basel Medizinische Fakultät		(1935) (1939)	81, 78
Universität Bern Medizinische Fakultät		(1938)	75

School	FAILED	Year Grad.	Number Failed
Medizinische Fakultät der Universität Wien		(1914), (1926)	2
Friedrich-Wilhelms-Universität Medizinische Fakultät, Berlin		(1923)	1
Schlesische-Friedrich-Wilhelms Universität Medizinische Fakultät, Breslau		(1923)	1
Universität Heidelberg Medizinische Fakultät		(1928)	1
Universität Basel Medizinische Fakultät		(1929)	1
Universität Zürich Medizinische Fakultät		(1905)	1

Forty-five physicians were successful in the practical examination for reciprocity and endorsement applicants held in Chicago, June 27. The following schools were represented:

School	PASSED	Year Grad.	Reciprocity with
University of Colorado School of Medicine		(1938)	Colorado
Rush Medical College		(1937)*	Missouri, (1938)* California
State University of Iowa College of Medicine		(1934), (1938)	Iowa
University of Louisville School of Medicine		(1935), (1939, 2)	Kentucky
Tulane University of Louisiana School of Medicine		(1936)	Louisiana
Univ. of Minnesota Medical School		(1932)	Wisconsin, (1938) Minnesota
St. Louis University School of Medicine		(1924), (1938), (1939),* (1939, 8)	Missouri
Washington University School of Medicine		(1937), (1939, 7)	Missouri
Creighton University School of Medicine		(1938)	Nebraska
University of Nebraska College of Medicine		(1936), (1937)	Nebraska
University of Cincinnati College of Medicine		(1918)	Ohio
Jefferson Medical College of Philadelphia		(1932)	Ohio
Vanderbilt University School of Medicine		(1937)*	Tennessee
University of Texas School of Medicine		(1938)	Texas
University of Virginia Department of Medicine		(1938)	Virginia

School	PASSED	Year Grad.	Endorsement of
Loyola University School of Medicine		(1937)	N. B. M. Ex.
Northwestern University Medical School		(1939, 2), (1940)	N. B. M. Ex.
The School of Medicine of the Division of the Biological Sciences		(1937)	N. B. M. Ex.
Tulane University of Louisiana School of Medicine		(1938)	N. B. M. Ex.
St. Louis University School of Medicine		(1938)	N. B. M. Ex.

* License has not been issued.

Texas Reciprocity Report

Dr. T. J. Crowe, secretary, Texas State Board of Medical Examiners, reports 102 physicians licensed to practice medicine by reciprocity and two physicians so licensed by endorsement on June 17-19 and July 28. The following schools were represented:

School	LICENSED BY RECIPROCITY	Year Grad.	Reciprocity with
Univ. of Arkansas School of Medicine		(1938, 2), (1939, 4)	Arkansas
College of Medical Evangelists		(1938)	Montana
University of Southern California School of Medicine		(1934)	California
Univ. of Colorado School of Medicine		(1931), (1938), (1939)	Colorado
College of Physicians and Surgeons of Chicago, School of Medicine of the University of Illinois		(1906)	Illinois
Loyola University School of Medicine		(1939)	Illinois
Northwestern University Medical School		(1935), (1937) (1937, 2)	Illinois, Michigan, (1938) Nebraska, (1939) California
Rush Medical College		(1930)	Missouri, (1933) Illinois, (1934) West Virginia, (1935) Minnesota
The School of Medicine of the Division of the Biological Sciences		(1937)	Michigan
University of Illinois College of Medicine		(1922), (1933)	Illinois
Indiana University School of Medicine		(1928), (1939)	Indiana
State University of Iowa College of Medicine		(1935)	Iowa
University of Kansas School of Medicine		(1929), (1939)	Kansas
University of Louisville School of Medicine		(1938)	Kentucky
Louisiana State University Medical Center		(1935)	Louisiana
Louisiana State University School of Medicine		(1939)*	Louisiana
Medical Department of Tulane Univ. of Louisiana		(1906)	Louisiana
Tulane University of Louisiana School of Medicine		(1929), (1932), (1936), (1937, 3), (1938, 3), (1939, 2)	Louisiana
Harvard Medical School		(1935)	New York
Tufts College Medical School		(1915) (1928)	Nebraska, New York, (1934) Maine
Detroit College of Medicine		(1930)	Michigan
University of Michigan Medical School		(1926), (1938)	Michigan
University of Minnesota Medical School		(1932)	Missouri
Ensworth Medical College		(1904)	Nebraska
St. Louis University School of Medicine		(1937), (1939)	Missouri
Washington University School of Medicine		(1933), (1935)	Missouri
Cornell University Medical College		(1934)	New York
New York University College of Medicine		(1938)	Louisiana
Syracuse University College of Medicine		(1928)	New York
Duke University School of Medicine		(1938)	N. Carolina
Ohio State Univ. College of Homeopathic Medicine		(1916)	Ohio
University of Cincinnati College of Medicine		(1936), (1937)	Ohio

Western Reserve University School of Medicine (1917), (1935), (1937) Ohio
University of Oklahoma School of Medicine (1930), (1936, 2), (1938, 2) Oklahoma, (1938) Maryland
Hahnemann Medical College and Hospital of Philadelphia ..... (1932) New Jersey, Pennsylvania
Jefferson Medical College of Philadelphia ..... (1894) Nebraska, (1921) Pennsylvania, (1932) Ohio
Temple University School of Medicine ..... (1938) Penna.
University of Pennsylvania School of Medicine ..... (1919) Penna.
Meharry Medical College ..... (1936), (1939) Tennessee
University of Tennessee College of Medicine .. (1936), (1937), (1938), (1939, 2) Tennessee
Vanderbilt University School of Medicine .... (1929), (1937) Tennessee
Baylor University College of Medicine ..... (1937) Washington
Medical College of Virginia ..... (1906) Arizona, (1931) Virginia, (1932) West Virginia
University of Virginia Department of Medicine ..... (1931) Virginia
McGill University Faculty of Medicine ..... (1902) New York
Rheinische Friedrich-Wilhelms-Universität Medizinische Fakultät, Bonn ..... (1934) New Jersey
Universität Köln Medizinische Fakultät ..... (1934) New York
University of Edinburgh Faculty of Medicine ..... (1932) New York
Universität Basel Medizinische Fakultät ..... (1935) New York
Osteopath † ..... California

LICENSED BY ENDORSEMENT

School	Year Grad.	Endorsement of
University of Oregon Medical School	(1933)	Alaska
Baylor University College of Medicine	(1939)	U. S. Navy

* This applicant has received the M.B. degree and will receive the M.D. degree on completion of internship.
† Licensed to practice medicine and surgery.

Book Notices

Arthritis and Allied Conditions. By Bernard I. Comroe, A.B., M.D., F.A.C.P., Instructor in Medicine, University of Pennsylvania, Philadelphia. Cloth. Price, $8.50. Pp. 752, with 200 illustrations. Philadelphia: Lea & Febiger, 1940.

Comroe's book is the most comprehensive English treatise on arthritis and allied conditions that has appeared in years. It is well printed and profusely illustrated.

The practitioner will appreciate the author's avoidance of the error of many previous writers, namely limiting the text to chronic arthritis. The arthritides of known and unknown etiology as well as many allied medical and orthopedic conditions are discussed. The diseases commonly mistaken for arthritis are listed and a synopsis of the more important clinical features of each one is presented.

The practitioner will find it a most usable and practical book containing the necessary information relative to the etiology, clinical course, diagnosis and prognosis of the various diseases of joints and many skeletal afflictions. The treatment of most of these conditions is given in detail. The more pertinent features of each disease are emphasized by presenting them in synopses which are set off by heavy black lines, thus making them readily available for the busy practitioner.

The objects of the book as outlined in the introduction have not been completely fulfilled. For his opinions the author has relied on the current literature, much of which has not been critically digested and summarized, rather than on clinical and laboratory experience. Had the book been better edited in this respect the reader would be saved considerable time and would be able to ascertain with more certainty Comroe's views on many controversial matters.

Two hundred and eighty-six pages pertain to rheumatoid arthritis; however, the extremely important subjects of degenerative joint disease, rheumatic fever and gout are dismissed with seventeen, twenty-four and twenty-eight pages respectively. Of these 286 pages only four are devoted to etiology, three to pathology and twenty to the clinical description of the disease; yet 125 pages are concerned with many therapeutic measures of questionable value. Adequate space is given, however, to the important subjects of physical therapy and orthopedic treatment. Many will question the necessity of devoting more than 200 pages to numerous allied orthopedic diseases or, if they approve, will wish that these subjects had been written by a competent, authoritative orthopedic surgeon and not an internist. The reviewer believes that much of this space should have been assigned to the other arthritides and to the many diagnostic problems which confront the student of diseases of joints. The subject of spondylitis, for example, is inadequately presented.

The book begins with a much abbreviated classification of joint diseases. The outline employed by the author in studying each new patient is presented and the important points to be looked for in the history and physical examination are stressed. The section on radiology in the diagnosis of the arthritides would have been strengthened by a more detailed description of the changes occurring at various stages. In addition is included a list of examinations frequently requested. Unfortunately, the diagnostic significance of these points and the indications for such tests are not given. Nor does he mention that many of the tests are expensive and that the information derived is of questionable merit.

The value of the sedimentation rate is discussed. Surprisingly, the author believes that "the results may be read directly without correction factors"! Little is said about the more accurate method devised by Rourke and Ernstene.

Much valuable information pertaining to the physiology of the articular structures is not included and the discussion of the origin and nature of normal synovial fluid is not complete. Many will disagree with the statement "There are no pathognomonic features of the chemical composition of synovial fluid in the various joint diseases." The cytologic and chemical characteristics of joint effusions when interpreted along with the clinical facts are often of diagnostic value.

The section on pathology of rheumatoid arthritis is unnecessarily short and incomplete. That rheumatoid arthritis nodules occur in other organs, including the eye (scleromalacia perforans), is not mentioned, although many of the visceral changes of this systemic disease are referred to.

From the section on etiology it is readily apparent that the author is an adherent of the infectious theory of the origin of rheumatoid arthritis. Before presenting any factual data he states that "an impartial evaluation of the evidence presented to date suggests that rheumatoid arthritis is probably some form of infectious disease." He fails to state that many of the clinical features which suggest that rheumatoid arthritis is an infectious disease are exhibited by gouty arthritis, a metabolic disease. The other etiologic theories of rheumatoid arthritis are not fully evaluated.

Despite the statement "Recently, a carefully conducted follow-up study has shown no permanent improvement in joint phenomena in a large series of patients with rheumatoid arthritis following careful and determined removal of all infected foci" the author burdens the reader with a twenty-five page dissertation on foci of infection in rheumatoid arthritis. The same section contains the statement that "there is a relationship between good gut function and arthritis but one cannot tell which is the cart and which the horse," yet in the following sentence he says "It is important to carefully check all of the factors in the patient's diet and to see that his colon functions well, but we do not know with any certainty what relation such hygienic measures bear to joint conditions." Such confusing statements are not helpful to the physician who seeks authoritative help. The book contains too many of them. A more critical evaluation of the subject matter contained in this section judged in the light of the author's personal experience would have strengthened the book greatly. As it stands, it is too controversial and indefinite to be helpful.

His clinical description of rheumatoid arthritis has obvious deficiencies and does not begin to compare with the detailed, scholarly dissertations of some of the older writers. Little is said concerning the many variations in onset and course of the disease nor are the diagnostic difficulties adequately discussed. He fails to stress that the differentiation of rheumatic fever from rheumatoid arthritis is often impossible without the passing of considerable time.

The sections dealing with the treatment of rheumatoid arthritis are complete. The subjects of physical therapy and orthopedic treatment are well covered and will be helpful. The chapter on diet is too detailed and unnecessarily long, considering that the author believes, as do many others, that the dietary of the patient with rheumatoid arthritis should be high in vitamins and calories (unless the patient is overweight) and adequate in iron, calcium and phosphorus. Although vitamin deficiencies are not specific for rheumatoid arthritis and not causally related, the author burdens the reader with fifteen pages on vitamins. It is most unfortunate that so many pages are devoted to such questionable therapeutic measures as sulfur, bee venom and

chaulmoogra oil, which are judged nonacceptable by many authorities, including the Council on Pharmacy and Chemistry of the American Medical Association. There exists no proof that any type of infectious disease can be cured by vaccines, yet Comroe advocates their use in rheumatoid arthritis, a disease of unknown etiology. He further confuses the issue by sanctioning their administration in degenerative joint disease, a noninfectious disease. Here again the book would have been strengthened had this questionable form of therapy been presented in a more decisive manner. Although endocrine disturbances have never been demonstrated to be of etiologic significance in rheumatoid arthritis, many pages are devoted to the subject. Despite the fact that most authorities have not observed beneficial results with roentgen therapy, Comroe unequivocably sanctions its use in the treatment of various forms of arthritis. In the case of gonococcic arthritis he evidently considers it as efficacious as fever therapy and the sulfonamide compounds! Again he fails to state what his personal experience with this form of therapy has been. Until it has been demonstrated that such measures are beneficial and without deleterious effect on articular cartilage, they should not be used indiscriminately. It is also difficult to ascertain what results the author has observed in rheumatoid arthritis patients treated with gold salts.

The subject of intermittent hydrarthrosis is not fully presented. The author fails to point out that careful follow-up studies will reveal that the majority of the cases are atypical forms of rheumatoid arthritis.

The information pertaining to spas will prove helpful to the practitioner desiring it. The section on case summaries contributes little to the book. If additional case reports, presented in more detail, had been included in the respective chapters, they would have served a useful purpose.

As mentioned before, the chief criticism of the book is that it is assembled from the literature and not written from the author's experience. In consequence Comroe's personal opinion concerning many controversial matters is never given. There is nothing constructive about the book and no evidence that the author has done any considerable amount of original work. All but six of the illustrations are borrowed from the literature The book would have been greatly improved, materially shortened and more readable had the subject matter (chiefly a digest of the current literature) been critically and concisely reviewed. Many of the references contained in the bibliographies are never referred to in the text. The absence of titles is therefore unfortunate, in that the reader cannot judge as to the content of many of the articles quoted. The index could be improved.

Despite these criticisms, the reviewer believes that this is the best book in English on the subject published in recent years. The student of joint diseases will wish it had been written by a more experienced authority and that the subject matter had been presented in a more scholarly manner.

Manson's Tropical Diseases: A Manual of the Diseases of Warm Climates. Edited by Phillip H. Manson-Bahr, C.M.G., D.S.O., M.D., Senior Physician to the Hospital for Tropical Diseases, London. Eleventh edition. Cloth. Price, $11. Pp. 1,083, with 397 illustrations. Baltimore: William Wood & Company, 1940.

The author continues to keep this book within the confines of a single handy volume. The provision of adequate discussions of so many complex diseases—kala-azar, plague, cholera, beriberi and other vitamin deficiency diseases, yellow fever, leishmaniasis, trypanosomiasis, malaria, yaws, leprosy, bilharziasis, rabies, typhus, undulant fever and numerous others—within one volume is a task which has been well done in this instance. Much ancient history has been deleted from the text and in its place are chapters on the preparation for life in the tropics, and a concise summary of the influence of the tropics in altering the facies of common diseases encountered in ordinary medical practice. Greater attention in this edition has been given to the subject of nutrition. Considerable revision has been made of the chapters on yellow fever, typhus and malaria, to make room for which the chapters on technic have been omitted. Mosquitoes, flies, fleas, worms, poisonous reptiles and fish, and the numerous microscopic causes, contribute toward making tropical diseases one of the most interesting subjects in medicine. This book emphasizes that fact very well.

Allergie und Tuberkulose: Allergische und verwandte Phänomene bei Tuberkulose. Von Dr. Franz Ickert, Oberregierungs- und Obermedizinalrat in Stettin. Boards. Price, 5.60 marks. Pp. 142, with 10 illustrations. Leipzig: Georg Thieme, 1940.

Because many observers utilize their own nomenclature and introduce new hypotheses and theories, discarding all prior conceptions so that it is difficult for the novitiate to compare different observations on allergy, particularly allergy in tuberculosis, Ickert presents in this small book the important allergic and related phenomena in tuberculosis freed as much as possible from the numerous theories and hypotheses. He subscribes to the Pirquet expression of 1906 "for the general conception of changed reaction ability I use the expression 'allergy.'" Under allergy in general he points out von Bergmann's observations of changed reactions in man to previous contact with mercury and iodine. In 1873 Blackley pointed to certain idiosyncrasies; in 1891 Koch demonstrated his "basic experiment"; Richet in 1902 produced "anaphylactic shock" and Arthus in 1903 produced the local subcutaneous or intracutaneous phenomenon considered an antigen antibody reaction resulting in the allergic reaction. The antigen antibody reaction does not occur in the blood or body fluids but in the cells, and this can be transferred passively (Prausnitz-Küstner 1922). Hyperergic inflammation possesses two phases, the exudative and the productive. The vegetative nervous system may play a part in the reactions. Certain so-called allergic diseases occur in man, probably tonus changes in the contractile elements and exudative reactions of the vascular connective tissue apparatus. In every tuberculous stage the existing allergy and the resistance of the organism as well as the amount of bacilli determine the form of tuberculosis, according to Ickert. The tuberculin reaction is dependent on the amount of tuberculin used and the height of hypersensitiveness. The tuberculin reaction is essentially specific but can display nonspecific effects. Many factors can influence its outcome. According to von Hayek, a high sensitivity is "hyperergy" and an abnormally low one "hypergy." "Normergy" exists with negative Pirquet and intracutaneous tests in the noninfected. "Euergy" exists with Pirquet negative, and intracutaneous reaction with 10^{-3} old tuberculin is "positive anergy"—the skin is able to destroy tuberculin. Nonspecific skin anergy exists under constitutional skin reactions or following certain diseases (measles). When the specific reaction power is so depressed that no reaction occurs to concentrated tuberculin, "dysergy" exists or "anergy" (of cachexia, starvation, old age).

The reactions in the various types of tuberculosis and related diseases are presented and elucidated by Ickert. Rössle considers (1) allergy with specific antigen antibody reaction, (2) parallergy occasioned by certain groups such as other infections and intoxications, and (3) pathergy, the reactions due to all other nonspecific irritants. Among constitutional influences on allergy are age, metabolism diseases such as diabetes and exophthalmic goiter, cardiovascular diseases, articular rheumatism, exudative lymphatic diatheses, and so on. When such diseases occur with tuberculosis, its outcome is determined by whether these diseases are related to syntropy or dystropy. The allergy acts as an accentuation factor of the syntropy or dystropy. Immunity and allergy as well as the Ranke teachings are given space, and finally desensitization is considered. In a final summarizing chapter Ickert points out that allergy is to be viewed as an independent biologic function. Thus also tuberculosis must be analyzed as a "changed reaction relation" of the organism. Each stage begins with an exudative phase which concludes with the specific granuloma, the tubercle. The former is associated with a high, the latter with a moderate allergic, hypersensitiveness. The height of the tuberculin threshold sometimes indicates the new tuberculosis stage earlier than the roentgenogram. The tuberculin and complement fixation reactions occur in reversed relation. Not only specific heightened hypersensitiveness but other sources of hypersensitiveness, such as other infections and focal toxicoses, exert an influence. Infections of all types occasion a disposition to such parallergic and pathergic reactions. On the other hand, an active tuberculous focus may act parallergic or pathergic toward other irritants. The disclosure of constitutional factors in individual cases may prove valuable (prognostically). Allergy in tuberculosis, as in some other diseases, assumes an important role which must not be forgotten—it leads us through analysis of the disease of an organ

to that of the disease of the individual and of the afflicted family for evaluation of man's entirety with relation to the family ancestry.

The book concludes with a good bibliography and a workable subject index of two pages. Although Ickert uses mainly German literature and expresses primarily his own views resulting from years of analysis, his conclusions are drawn rationally and are worth consideration by all interested in the practical field of allergy, especially as it pertains to tuberculosis in its various phases and as it is related to other diseases. The ten illustrations, mostly x-ray films, are well presented. This small textbook is in German and can be recommended for the personal as well as the tuberculosis and medical library.

A New Dictionary of Chemistry. Edited by Stephen Miall, LL.D., B.Sc. With the Assistance of Many Well-Known Chemists. Cloth. Price, $15. Pp. 575. New York, Toronto & London: Longmans, Green & Company, 1940.

Chemical science is advancing rapidly. The additions to chemistry and the related sciences have led to new methods of description and designation. To clarify this complexity the author, assisted by many well known chemists, has compiled a single volume of definitions and brief discussions of chemical terms the keynote of which is conciseness. This is not only a dictionary of chemistry but a great source of accurate chemical information conveniently arranged. The chemical elements are each included in a separate entry and the entire series is reviewed under such headings as the elements and their electrons, isotopes and the periodic table. Some compounds of the elements are also surveyed and their crystal structures as revealed by x-ray analysis are indicated by diagrams. There is included also a roll of notable chemists of the present and past generations, with the more important achievements of each individual. The dictionary is intended to be useful not only to chemists but to many others—doctors, druggists, chemical manufacturers, lawyers, journalists—who desire to refresh their memories about a subject which they have partly forgotten.

Anatomy and Physiology Laboratory Manual and Study Guide. By Barry Griffith King, Ph.D., Assistant Professor of Physiology, College of Physicians and Surgeons, Columbia University, New York, and Helen Maria Roser, B.A., R.N., Instructor in Nursing, Department of Nursing, College of Physicians and Surgeons, Columbia University, New York. With a foreword by Isabel M. Stewart. Paper. Price, $2.75. Pp. 273, with 63 illustrations by M. Cartwright Baker. Philadelphia & London: W. B. Saunders Company, 1940.

This manual was prepared specifically for use in schools of nursing but would be applicable to college courses as well. It is based on the experience of the authors in actual laboratory work. The loose leaf arrangement is similar to other laboratory manuals in use. The division and sequence of subject matter are logical and well integrated. Many of the questions asked appear to be rather too simple for stimulating thought and might be characterized as "leading questions." On the other hand, many of the exercises call for independent observation and initiative. Directions for dissection are well illustrated with original sketches. An admirable feature is the large number of experimental observations that the students are required to make with themselves or mates as subjects. Throughout there is good integration of anatomy and physiology. In the appendix are several pages of descriptive and explanatory notes which should greatly simplify the work for those unfamiliar with laboratory equipment and technic.

Man Alive You're Half Dead! A Physician Tells You How to Eat Your Way to Glowing Health . . . and Stay There. By Daniel Colin Munro, M.D., Medical Director of the Lake Placid Club, New York. Cloth. Price, $2. Pp. 209. New York: Dudley Courtenay, Publisher, 1940.

On the basis of many undisputed facts this book promulgates in a slightly modified form the typical fallacies of food faddism, including the compatible eating fad and such unsupported theories as that coronary thrombosis is most commonly caused by deficiency of vitamin C. Excessive emphasis is placed on the unestablished belief that gelatin is valuable for "revitalizing those who are chronically weak and tired." The necessity for taking yeast and vitamin concentrates is excessively emphasized. The book cannot be classed among those which can be recommended to lay inquirers seeking authentic information about diet and health.

Report on the Sex Question. By The Swedish Population Commission. Translated and edited by Virginia Clay Hamilton, M.D. Statens Offentliga Utredningar 1936: 59, Stockholm. Published for National Committee on Maternal Health, Inc. Cloth. Price, $2. Pp. 182. Baltimore: Williams & Wilkins Company, 1940.

This is a translation and edited edition of a report of the Swedish Population Commission. It offers an analysis of the use of contraception in Sweden, giving at the same time a study of the relationship of contraception to housing, nutrition, health and innumerable other aspects of life. It discusses various technics of contraception and reaches the conclusion that the combination of ointment and pessary, which are wholly under the control of the female, represents the preferred technic. It recognizes also, however, that all technics are subject to error and that the ideal is not yet available. There is a discussion as well of the problems of the control of venereal disease and of sex enlightenment. The commission is inclined to favor the use of any type of mechanical or chemical effort that will prevent the transmission of venereal disease and believes in sex enlightenment applied through schools and through the home in order to secure more widespread use of such methods. As a result of this report a number of measures were passed by the 1937 Swedish legislative body, including maternity aid in the form of cash, aid for orphan children, loans for the purchase of homes or furniture for engaged and newly married couples, modification of the school curriculum to include sexual enlightenment and repeal of the old Swedish contraceptive law.

Chambers's Technical Dictionary Comprising Terms Used in Pure and Applied Science: Medicine: the Chief Manufacturing Industries: Engineering: Construction: the Mechanic Trades. With Definitions by Recognized Authorities. Edited by C. F. Tweney and L. E. C. Hughes, A.C.G.I., D.I.C., B.Sc. Cloth. Price, $5. Pp. 957. New York: Macmillan Company, 1940.

The need for an adequate dictionary of technical terms in different fields is readily apparent. This dictionary is aimed at giving "definitions of terms that are of importance in pure and applied science, in all branches of engineering and construction, and in the larger manufacturing industries and skilled trades." The commoner medical terms are reasonably well covered, but many of the more technical ones such as "adiadokocinesis" and "agoraphobia" do not appear. This is not said by way of criticism, since there would ordinarily be little use in including the rarer terms of this nature in a general technical dictionary. In general this dictionary may be expected to serve a useful purpose which subsequent editions may enhance.

Observations Made During the Epidemic of Measles on the Faroe Islands in the Year 1846. By Peter Ludwig Panum, M.D. Translated from the Danish by Ada Sommerville Hatcher. With a Biographical Memoir by Julius Jacob Petersen, M.D. Translated from the Danish by Joseph Dimont. Introduction by James Angus Doull, M.D. Published by the Delta Omega Society. Cloth. Price, $2.50. Pp. 111, with portrait. New York: American Public Health Association, 1940.

This book is published by the Delta Omega Society in memory of Wade Hampton Frost, epidemiologist. It is the third public health classic republished by the society, the first being Budd's Typhoid Fever, the second Snow's Cholera. In his introduction the editor, J. A. Doull, describes the conditions under which Panum worked and analyzes ably the significance of his great contribution to the epidemiology of measles. Then comes a translation by Joseph Dimont of Julius J. Petersen's highly interesting sketch of Panum's career and personality (*Nord. med. Ark.* **17**:1 [No. 24] 1885). The translation of Panum's Observations was made by Ada S. Hatcher in 1924 and was published in Medical Classics, 1939. The present edition has been revised and corrected. "Efforts have been made to be as literal as is consistent with an intelligent rendering and to catch in the translation something of the quaintness of Panum's style." In appendixes are notes by the editor and the translators as well as translations of two other Danish reports on the 1846 Faroe measles epidemic. By his observations on the spread of measles in the Faroe Islands in 1846, recorded with wonderful clarity and precision, Panum made a "contribution to epidemiology which will stand for all time." The republication in English translations of Panum's report and of Petersen's sketch of Panum is of interest to all students of infectious epidemiology and of medical history.

Bureau of Legal Medicine and Legislation

MEDICOLEGAL ABSTRACTS

Hospitals: Criteria of Charitable Character for Tax Purposes.—The R. B. Smith Memorial Hospital Association, located at Alma, Mich., was organized as a nonprofit corporation, the community of Alma subscribing approximately $24,000 to equip it. According to the evidence, although the hospital association made a book charge against every patient who received its services, it attempted collection only from those actually able to pay in full or in part. County patients and afflicted children were treated at less than cost, and indigents were treated without cost, the uncollected accounts of such patients amounting to approximately $3,500. During certain periods revenue exceeded expenditures, while in others it did not. Whenever a deficit resulted it was met by further donations from the community. On those few occasions when there was no deficit, the surplus was used to finance new equipment or additions to the hospital. No dividends had ever been paid to, or expected by, the members of the hospital association. The auditor general of Michigan, contending that the defendant association was not exempt from the real property tax as a charitable organization, petitioned for the sale of certain lands for taxes assessed against the association. From the judgment of the trial court holding that the defendant was exempt from taxation, the auditor general appealed to the Supreme Court of Michigan.

The definition of a charitable institution for purposes of tort law, said the Supreme Court, is not conclusive in a tax exemption case, in which case the exemption is to be construed strictly. As was held in *Michigan Sanitarium & Benevolent Ass'n* v. *City of Battle Creek*, 138 Mich. 676, 101 N. W. 855, for tax purposes a corporation is sufficiently charitable to entitle it to the privileges of exemption when the charges collected for its services are not more than are needed for its successful maintenance. Under this doctrine, in the opinion of the court, the defendant was clearly entitled to exemption from taxation. The auditor general's argument that the defendant should be tested each year, apparently on the theory that it would be subject to a tax in those years in which it operated at a profit, did not meet with the court's approval. Such a procedure would be absurd. By bobbing back and forth and calling the defendant tax exempt one year and not exempt another year, "all sorts of trouble" would result. The real test is what is the situation from year to year and is the hospital association operated for the benefit of its members or for the benefit of the community, in which case it would be a charitable organization. In the judgment of the court, the defendant hospital association operated a public and not a private hospital. The hospital was operated "without gain, profit or advantage to any one." The association did not lose its charitable character even though in some years, instead of the usual deficit, it showed a small surplus which it utilized to supply needed equipment. Judgment for the defendant was therefore affirmed.—*Gundry, Auditor Gen. v. R. B. Smith Memorial Hospital Ass'n (Mich.), 291 N. W. 213.*

Malpractice: Liability of Physician for Failure to Take Roentgenograms Before Attempting to Remove Broken Needle.—The defendant, a specialist in diseases of the ear, nose and throat, was engaged to remove the plaintiff's tonsils. His assistant, while injecting procaine hydrochloride into the tonsillar area of the plaintiff's throat, broke off the point end of a hypodermic needle which became embedded in the tissues of plaintiff's throat. The assistant called the defendant and informed him of the mishap. After a physical examination of the plaintiff's throat it was decided that the broken needle might be embedded in the tonsil and that it would be best to proceed with the removal of that organ. This was done, but the broken needle was not contained in the removed tonsil. The defendant then made two attempts, for periods of forty-five minues and twenty-five minutes respectively, to remove the broken needle by probing from the inside, but he was unsuccessful. He then sent the plaintiff home with instructions to return to his office in about ten days. On the patient's return, the defendant caused roentgenograms to be taken and made another unsuccessful attempt to remove the broken needle. So far as the report shows, the needle was never removed in spite of attempts by other physicians to do so. The plaintiff later brought suit for malpractice against the defendant and obtained a judgment in his favor in the trial court. This judgment, however, was reversed by the supreme court of New York, appellate division, second department, and the plaintiff appealed to the Court of Appeals of New York.

The plaintiff did not claim that the defendant or his assistant was negligent in breaking the needle. He did contend, however, that the defendant was negligent in failing to take roentgenograms immediately after the mishap to determine the location of the broken needle and in futilely probing for the needle when such procedure would likely push it still farther into the tissues of the plaintiff's throat. At the trial, "while there was strong evidence to the contrary," the plaintiff's expert medical witness, also a specialist in diseases of the ear, nose and throat, testified that under the circumstances it was not proper medical practice for the defendant to proceed with the tonsillectomy; that, since a broken needle does not remain stationary but is immediately sucked into the muscles, it was not good practice to attempt to remove the broken needle without first having taken roentgenograms and located the position of the fragment, and that it was folly for the defendant to attempt to remove the fragment by probing because the only practical way to remove it was from the outside through an incision in the neck. He would not say that there was no possibility of success in the method employed by the defendant "because coincidence plays a big part." In his opinion, the defendant's operation was "a departure from approved methods in general use" at the time.

The testimony of the plaintiff's expert witness, said the Court of Appeals, was not merely to the effect that the witness would have treated the patient differently but rather that the method adopted by the defendant was not proper and approved practice. In the opinion of the court, the witness's testimony was not nullified, as the supreme court believed, by his admission that probing might have been successful. By so testifying the witness merely allowed for coincidence. His testimony, in effect, was that the defendant might have gotten hold of the broken needle by probing from the inside but that the proper and approved practice was to remove it from the outside. The court concluded that, while the evidence in favor of the defendant was strong and on the weight of the evidence the result reached by the supreme court may have been correct, it cannot be said that there was no evidence to support the jury's verdict for the plaintiff. Accordingly, the judgment of the trial court in favor of the plaintiff, which the supreme court had reversed, was reinstated.—*Bernstein v. Greenfield (N. Y.), 22 N. E. (2d) 242.*

Society Proceedings

COMING MEETINGS

American Academy of Pediatrics, Memphis, Tenn., Nov. 18-20. Dr. Clifford G. Grulee, 636 Church Street, Evanston, Ill., Secretary.

American Society of Anesthetists, New York, Dec. 12. Dr. Paul M. Wood, 745 Fifth Ave., New York, Secretary.

American Student Health Association, Ann Arbor, Mich., Dec. 27-28. Dr. Ralph I. Canuteson, University of Kansas, Lawrence, Kan., Secretary.

Eastern Section, American Laryngological, Rhinological and Otological Society, Philadelphia, Jan. 10. Dr. N. S. Weinberger, Robert Packer Hospital, Sayre, Pa., Chairman.

Puerto Rico, Medical Association of, San Juan, Dec. 13-15. Dr. David E. Garcia, P. O. Box 3866, Santurce, Secretary.

Radiological Society of North America, Cleveland, Dec. 2-6. Dr. Donald S. Childs, 607 Medical Arts Bldg., Syracuse, N. Y., Secretary.

Society of American Bacteriologists, St. Louis, Dec. 27-29. Dr. I. L. Baldwin, Agricultural Hall, University of Wisconsin, Madison, Wis., Secretary.

Southern Section, American Laryngological, Rhinological and Otological Society, Nashville, Tenn., Jan. 8. Dr. William G. Kennon, Doctors Bldg., Nashville, Tenn., Chairman.

Southern Surgical Association, Hot Springs, Va., Dec. 10-12. Dr. E. Alton Ochsner, 1430 Tulane Ave., New Orleans, Secretary.

Southwestern Medical Association, Tucson, Ariz., Nov. 21-23. Dr. M. P. Spearman, 1001 First National Bank Bldg., El Paso, Texas, Secretary.

Western Surgical Association, Topeka, Kan., Dec. 6-7. Dr. Albert H. Montgomery, 122 South Michigan Blvd., Chicago, Secretary.

Current Medical Literature

AMERICAN

The Association library lends periodicals to members of the Association and to individual subscribers in continental United States and Canada for a period of three days. Three journals may be borrowed at a time. Periodicals are available from 1930 to date. Requests for issues of earlier date cannot be filled. Requests should be accompanied by stamps to cover postage (6 cents if one and 18 cents if three periodicals are requested). Periodicals published by the American Medical Association are not available for lending but can be supplied on purchase order. Reprints as a rule are the property of authors and can be obtained for permanent possession only from them.

Titles marked with an asterisk (*) are abstracted below.

American Journal of Ophthalmology, St. Louis

23:971-1088 (Sept.) 1940

Retinal Lesions in Lupus Erythematosus. A. E. Maumenee, Baltimore.—p. 971.
Concerning the Chamber Angle: II. Exfoliation of Zonular Lamella and Glaucoma Capsulare. H. S. Gradle and H. S. Sugar, Chicago.—p. 982.
Retinal Changes with Marked Impairment of Vision in Measles: Report of Case. F. R. Shlossberg and M. Prizer, Haverhill, Mass.—p. 998.
Inherited Eye Defect in the Guinea Pig: Report of Further Anatomic Studies. H. L. Foust, Ames, Iowa.—p. 1000.
Treatment of Sequels of Perforated Corneal Ulcer: Note. P. C. Kronfeld, Chicago.—p. 1014.
Management of Some Complications Which Follow Cataract Extraction. E. B. Spaeth, Philadelphia.—p. 1019.
Conjunctival Lesions in Tuberous Sclerosis. T. H. Luo, Peking, China.—p. 1029.

Am. J. Roentgenol. & Rad. Therapy, Springfield, Ill.

44:325-484 (Sept.) 1940

Abnormally Wide Respiratory Movement of Lower Lung Structures: Roentgen Evidence of Obstructive Emphysema. R. Golden, New York.—p. 325.
*Roentgenologic Aspects of Epidemic of Acute Respiratory Tract Infection. K. Kornblum and H. A. Reimann, Philadelphia.—p. 333.
Roentgen Aspects of Nonputrid Pulmonary Suppuration. M. L. Sussman, New York.—p. 345.
Basal Exudates of Subphrenic Origin. L. R. Sante, St. Louis.—p. 350.
Primary Carcinoma of Lung: Roentgenologic Study of 206 Proved Cases. J. W. Olds and B. R. Kirklin, Rochester, Minn.—p. 357.
Recent Progress in Bronchographic Examination of Bronchogenic Carcinoma. P. L. Fariñas, Havana, Cuba.—p. 370.
Roentgenologic Observations in Leprosy. M. J. Oberdoerffer and D. R. Collier, Chiengmai, Thailand.—p. 386.
Xanthomatosis (Lipoidosis, Schüller-Christian Type): Report of Case. D. S. Kellogg, Fort Sam Houston, Texas.—p. 396.
Effect of Fractional Roentgen Irradiation on Several Groups of Inoperable Tumors. C. Fried, São Paulo, Brazil.—p. 401.
Debt of Radiology to the Thomsons and the Thompsons, Physicists and Engineers: Historical Memoir. P. Brown, Egypt, Mass.—p. 409.
Principles of Relationship Between Hospitals and Radiologists. B. R. Kirklin, Rochester, Minn.—p. 423.
Measurement in Roentgens of Gamma Radiation from Radium by Free Air Ionization Chamber. L. S. Taylor and G. Singer, Washington, D. C.—p. 428.
Further Studies on Role Played by Certain Normally Occurring Intracellular Chemical Compounds in Growth and Development. S. P. Reimann, Philadelphia.—p. 444.

Roentgenologic Aspects of Epidemic of Acute Respiratory Tract Infection.—Kornblum and Reimann present x-ray data of eighty-six patients who suffered from acute epidemic infection of the respiratory tract which occurred in January 1939. This study was prompted by the fact that from two patients in a small series of similar cases which occurred sporadically during 1938 a filtrable infectious agent was recovered and that the 1939 outbreak might be due to the same etiologic agent. The patients were medical students, nurses and interns of the Jefferson Medical College and Hospital, making a total of 813 persons, 407, or 50 per cent, of whom at some time between January and May 1939 were ill with the disease. Of the 407 patients, about 100 required hospital care, and of these eighty-six were studied roentgenologically. There were no fatalities, indicating the relatively mild character of the disease. The condition was primarily an inflammation of the mucous membrane of the respiratory tract, occasionally including the trachea and bronchi, and in a few instances the bronchioles and lungs. The majority of the patients (87 per cent) were ill with nasopharyngolaryngitis, 7 per cent had in addition tracheobronchitis and 6 per cent had tracheobronchopneumonia. The disease with pneumonia lasted from two to seventeen days, averaging 8.2 days. The disease was considered a distinct clinical entity similar to epidemic influenza but caused by a different agent. Many features of the disease suggest that a filtrable virus is the cause. According to the x-ray signs, of the eighty-six patients forty were normal, twenty-four had an acute tracheobronchitis and twenty-two had an atypical pneumonia. There was a striking agreement between the clinical and the x-ray observations. Only four patients considered negative roentgenologically were clinically diagnosed as having acute tracheobronchitis. Two patients with an x-ray diagnosis of acute tracheobronchitis were clinically considered to have pneumonia. Roentgenologically no diagnostic difficulties were encountered in the recognition of the normal persons and those with pneumonia (atypical). The diagnosis of acute tracheobronchitis afforded considerable opportunity for error, as the general appearance is similar to that found in the chronic condition except that the shadows are less well defined, present a distinctly blurred appearance, and are less dense and of a softer character, and often there is small soft mottling in the pulmonary tissues about the hilus area. Of considerable importance is the generalized haziness throughout the lung field, giving the impression of an underexposed roentgenogram. This is evidently the result of an active congestion of the interstitial blood vessels throughout the lungs. The lateral view of the chest was of considerable value in recognizing acute tracheobronchitis and in differentiating it from the chronic variety. The pulmonary changes in the twenty-two patients were sufficient to warrant the diagnosis of pneumonia. A distinctive feature was a well localized but not large area of increased density, usually in the lower lobes of the lung. Although localized, the inflammatory reaction was not sharply defined. The inflammatory process spread and sometimes became bilateral. Another common lesion was a diffuse but not very dense pneumonic process at one base. The condition frequently became bilateral. In several cases the pneumonia showed a predilection for the hilus region, either unilateral or bilateral, presenting an x-ray appearance similar to the pneumonia seen in epidemic influenza. At times the pneumonic reaction was indicated by a barely perceptible haziness, its recognition depending on the diffuse unilateral or bilateral mottling in the involved area. As most of the patients were examined early, x-ray changes were evident in several cases before physical signs warranting a clinical diagnosis of pneumonia appeared, thus suggesting the probable development of pneumonia in a certain location. The x-ray changes tended to persist for some time after the apparent recovery of the patient. No pulmonary or pleural complications occurred and all the patients recovered.

American Review of Tuberculosis, New York

42:297-430 (Sept.) 1940

*Pathogenesis of Bronchiectasis. F. Fleischner, Boston.—p. 297.
Meningeal Blood Vessels in Tuberculous Meningitis. N. W. Winkelman and M. T. Moore, Philadelphia.—p. 315.
Carcinoma and Tuberculosis of Lung. C. E. Hamilton and N. H. Wexler, Brooklyn.—p. 334.
Tuberculosis in Rabbits: Further Comparison of Roentgenologic and Pathologic Findings in Primary and Reinfection Experimental Pulmonary Tuberculosis in Rabbits: Part 1. Primary Tuberculosis. H. E. Burke, Ray Brook, N. Y.—p. 343.
Urinary Iodine in Pulmonary Tuberculosis. K. P. Klassen, G. M. Curtis and R. A. Hancock, Columbus, Ohio.—p. 376.
Epidemiologic Trends of Tuberculosis. M. Pinner, New York.—p. 382.
Technic of Cavity Aspiration (Monaldi). E. Kupka and Ruth Wells, Olive View, Calif.—p. 401.

Pathogenesis of Bronchiectasis.—Fleischner reports three cases of benign bronchial tumors which seem to contribute to the knowledge of the pathogenesis of bronchiectasis. The three cases are regarded as different developmental stages of the same disease. In the first case, the earliest stage, there is a stenosis and beyond it obstructive emphysema without other gross pathologic pulmonary or bronchial changes. The third case represents a late stage with complete collapse of the lobe beyond the stenosis and marked purulent bronchiectasis. In the second case the growing intrabronchial tumor apparently caused a collapse of the left lower lobe with dry bronchiectasis. A superimposed purulent bronchitis led to its discovery. Pathogenically the second case is placed between the first and the last. These cases seem to demonstrate the underlying causative factor of bronchiectasis with the clearness of an experiment. In the beginning there is bronchial stenosis without other changes.

It impairs the air current in the affected lobe. The consequences of this disturbed ventilation to the lung are manifold and intricate, even if the interference with gas exchange is omitted. The decrease in ventilatory exchange means a decrease in respiratory plasticity of the lung and this raises difficulties in the spatial adjustment during the alternating phases of respiration. The next step is retention of secretion in small bronchi and bronchioles, as ineffective hawking and coughing due to the deficient respiratory air current decrease the self-cleansing capacity of the lung. The retained secretion will plug small bronchi, and atelectasis of the obstructed portions of parenchyma may ensue. Concomitant infections of the respiratory tract may intensify this series of events. Stenosis of a main or lobar bronchus or from an aspirated foreign body represents only one of several possible occurrences which reduce the ventilation. The occlusion of small peripheral bronchi and bronchioles appears to be a further indispensable link in the pathogenesis of bronchiectasis. In acute infections, chronic lesions of the respiratory tract and general or localized impairment of respiratory efficiency, bronchial occlusions occur which eventually lead to circumscribed atelectasis. Further decisive factors are the size of the involved area, the kinds and degrees of mechanical hindrances, the ability of the adjacent structures to perform the special adjustment and the rate of the primary collapse and the engorgement, the speed of the resorption of this edema and the period of time over which the atelectasis persists. The author conceives the development of bronchiectasis to be as follows: By diminution of the respiratory efficiency of a lung, a lobe or a portion of a lobe, the ability to get rid of secretion is impaired, the physiologic secretion is inadequately removed and small bronchi are blocked, or bronchi may be blocked because of an increased amount of secretion owing to inflammation, swelling of mucous membranes, granulation tissue or scars. These changes, singly or together, produce the conditions of preatelectasis and atelectasis. Stiffening and contraction of the tissue effect a pathologically increased pull on the bronchial wall, thus expanding the bronchus. The dilatation is first reversible but later becomes a permanent one if the intervening factors last for a long time. Atelectasis hampers the removal of secretion and causes inefficient ventilation of the neighboring parenchyma. Thus the condition becomes chronic and involves the surrounding normal portions. The inflammation with ensuing weakening of the bronchial wall may favor the development of bronchiectasis.

Archives of Dermatology and Syphilology, Chicago

42:543-754 (Oct.) 1940

Cultural Aspects of Dermatologic Thought. F. C. Knowles, Philadelphia. —p. 543.
Treatment of Acne Vulgaris by Cryotherapy (Slush Method). W. L. Dobes and H. Keil, New York.—p. 547.
Late Syphilis with Rare Orbital Gumma: Report of Case. H. R. Sniderman, Toronto, and E. A. Glicklich, Cincinnati.—p. 559.
Black Hairy Tongue: Report of Three Cases. C. B. Kennedy and J. K. Howles, New Orleans.—p. 566.
Xeroderma Pigmentosum in Negress. H. King and C. M. Hamilton, Nashville, Tenn.—p. 570.
Treatment of Dermatophytosis Pedis with Medicated Insoles. M. Seldowitz, Brooklyn.—p. 576.
Generalized Herpes Zoster Associated with Leukemia. U. J. Wile and H. H. Holman, Ann Arbor, Mich.—p. 587.
Acne Vulgaris: Review of Histologic Changes Observed in Early Lesions. F. W. Lynch, St. Paul.—p. 593.
Lichen Planus of Nail Bed: Report of Case. G. M. Lewis, New York, and J. F. Ricchiuti, Mahanoy City, Pa.—p. 607.
Microsporosis of Scalp Caused by Microsporum Fulvum: Report of Case and Description of Fungus. M. Moore and A. H. Conrad Jr., St. Louis.—p. 610.
Extensive Lichenified Eruption Caused by Trichophyton Rubrum. J. H. Swartz, Boston, and N. F. Conant, Durham, N. C.—p. 614.
Micropapular Tuberculid. C. W. Laymon and H. E. Michelson, Minneapolis.—p. 625.
*Arsenic as Cause of Cancer of Mucous Membrane: Report of Case. W. H. Goeckerman and L. F. X. Wilhelm, Los Angeles.—p. 641.
*Treatment of Chancroid with Sulfanilamide. O. Canizares, New York, and J. A. Cohen, Brooklyn.—p. 649.
Familial Lichen Planus: Report of Four Cases of Lichen Planus in One Family, with Brief Review of Literature. M. H. Saffron, Passaic, N. J.—p. 653.

Arsenic as Cause of Mucous Membrane Cancer.—Goeckerman and Wilhelm report a case in which common cutaneous lesions produced by arsenic were present and following its use the metal was demonstrated by microchemical studies in a papilloma and a carcinoma of the mucous membranes of the ureter and bladder, indicating that arsenic was at least a factor in their production. The patient presented classic arsenical keratoses of the palms of the hands and the soles of the feet. The keratoses of the trunk and extremities were of the arsenical rather than of the superficial epitheliomatosis type. Some of the keratoses suggested Bowen's disease. A section of a simple keratotic lesion of the trunk showed dermatitis and the presence of arsenic crystals in moderate amount, which is not necessarily of etiologic significance. In the potentially malignant ureteral papilloma, arsenic was demonstrated in a moderate amount and in the vesical papillomas in a considerable amount. The authors believe that their case is the only one of its kind so far reported suggesting that either cancer of the mucous membranes due to hematogenous distribution of arsenic is rare or the subject has not been investigated sufficiently.

Treatment of Chancroid with Sulfanilamide.—Canizares and Cohen present data on the comparative results of treating chancroid with sulfanilamide and local compresses soaked in a 1 to 5,000 solution of potassium permanganate and of iodoform powder and intravenous injections of Ducrey and typhoid vaccine. The authors considered the action of vaccine to be nonspecific. Patients treated with sulfanilamide had local dressings wet only with saline solution. The usual daily dosage of sulfanilamide was 75 grains (5 Gm.) divided into three doses for three days and 45 grains (3 Gm.) daily thereafter. Toxic symptoms were relatively uncommon. Sixty-four of ninety-three patients with lesions classified as small chancroids received sulfanilamide. The average time required for cure was twelve days. The condition in the twenty-nine control patients disappeared in an average of 11.6 days. Local therapy failed to control the lesions of four patients and these were later controlled by another type of local treatment. This never happened with sulfanilamide. Of sixty-eight patients with large chancroids, thirty-eight received the drug and required eighteen days for cure. The lesions of the thirty control patients healed in 22.7 days. Two of the sulfanilamide group and one of the control relapsed. Of seventeen patients with phagedenic chancroids, six control patients were cured in an average of thirty-five days. The lesions of two relapsed. Six of the eleven patients treated with sulfanilamide were healed in 27.6 days. However, the remaining five did not respond to this form of therapy, necessitating a change to iodoform powder followed by cure. Of the patients with inguinal adenopathies there were thirty-nine with unruptured and twelve with ruptured buboes. Rest in bed was given until the acute inflammatory symptoms had subsided. The buboes were aspirated whenever this was required. Local therapy (wet dressings, ice bags, the injection of iodoform in glycerin and vaccinotherapy) accomplished the desired effect in sixteen days in twenty of the unruptured cases and in nineteen days in the nineteen cases of the sulfanilamide series. This is an evident lack of superiority of the drug in this manifestation. Four control patients with ruptured buboes healed in an average of fifty-one days. The remaining eight received sulfanilamide and were cured in twenty-one days. The authors conclude that there is no advantage in giving sulfanilamide to patients with small and uncomplicated chancroids. Local therapy seems to be sufficient. If prompt control is not obtained, sulfanilamide is to be given. For large chancroids, sulfanilamide associated with local therapy should be used. In phagedenic chancroid it should be tried in conjunction with local therapy. Sulfanilamide does not decrease the formation of pus. It is most efficacious for ruptured buboes. Sulfanilamide should not be used in a routine manner but with knowledge of its value and indications.

Archives of Physical Therapy, Chicago

21:513-576 (Sept.) 1940

Evolution of Physical Therapy in Medicine. N. H. Polmer, New Orleans.—p. 517.
Effect of Cold, Heat and Weather on the Human Being. W. F. Petersen, Chicago.—p. 522.
Fever Therapy by Physical Means in Dermatology. K. Phillips and A. B. Litterer, Miami, Fla.—p. 533.
Hysterosalpingography a Deciding Factor in Management of Female Pelvic Pathology. G. Lyford and B. Billman, Cincinnati.—p. 541.
Radium and X-Rays in Treatment of Cancer of Head and Neck. G. S. Sharp, Pasadena, Calif.—p. 549.

Arkansas Medical Society Journal, Fort Smith

37:93-112 (Oct.) 1940

Surgical Relief of Pain. J. J. Keegan, Omaha.—p. 93.
Cesarean Section. C. R. Henry, Little Rock.—p. 96.

Florida Medical Association Journal, Jacksonville

27:113-164 (Sept.) 1940

Infection of Nasal Accessory Sinuses in Children. W. W. Quillian, Coral Gables.—p. 127.
Otologic Progress. M. A. Lischkoff, Pensacola.—p. 130.
Water Metabolism. E. P. Preston, Miami Beach.—p. 133.
Isolated Myocarditis. E. C. Chamberlain, Fort Lauderdale.—p. 137.
Inguinal Hernia: Analysis of 204 Operations. D. C. Robertson, Orlando.—p. 140.
Kidney Infections as Result of Obstruction. C. F. Bowie, Leesburg.—p. 144.
Venereal Disease Control. G. F. Highsmith, Arcadia.—p. 147.

Georgia Medical Association Journal, Atlanta

29:429-474 (Sept.) 1940

Sulfanilamide and Its Derivatives. E. A. Allen, Atlanta.—p. 429.
Treatment of Cardiac Edema. E. A. Bancker Jr., Atlanta.—p. 436.
Infantile Diarrhea. C. D. Fowler, Atlanta.—p. 442.
Use of Nicotinic Acid in Pellagra and Other Conditions. A. E. Siegel, Macon.—p. 447.
Intensive Fractional Treatment of Early Syphilis: Preliminary Report. J. S. Howkins, Savannah.—p. 452.
The Good of Yesterday. R. S. Leadingham, Atlanta.—p. 455.

Iowa State Medical Society Journal, Des Moines

30:465-508 (Oct.) 1940

*Acoustic Neuroma Producing Tic Douloureux. W. D. Abbott and B. M. Merkel, Des Moines.—p. 465.
Generalized Itching. R. Nomland, Iowa City.—p. 468.
Headache. J. C. Parsons, Des Moines.—p. 470.
Backache: Medical Phases. H. W. Rathe, Waverly.—p. 473.
Id.: From Orthopedic Standpoint. A. F. O'Donoghue, Sioux City.—p. 476.
Backache as Seen by a Gynecologist. W. F. Mengert, Iowa City.—p. 480.
Indigestion and Abdominal Pain. W. D. Paul, Iowa City.—p. 482.
X-Ray Anemia and X-Ray Death of Small Animals. M. C. Terry, Knoxville, and S. B. McFarland, San Francisco.—p. 486.
Appendicitis Deaths in 705 Necropsies. F. P. McNamara and R. V. McKay, Dubuque.—p. 487.

Acoustic Neuroma Producing Tic Douloureux.—According to Abbott and Merkel, only eighteen cases have been reported in which neuroma of the acoustic nerve produced typical pain resembling that of major trigeminal neuralgia. A woman aged 30 was referred to one of the authors because of pain in the right upper lip, nose and forehead. The pain was sharp and lancinating and was induced by touching the upper lip, talking or eating. Examination was negative except for a persistent trigger zone in the right upper lip; touching this area produced pain in the first and second branches of the right trigeminal nerve. Inhalations of trichlorethylene were tried temporarily. The pain became more intense and alcohol injection of the first and second branches of the right trigeminal nerve was performed. This freed the patient from pain for about seven months, after which pain returned with the same radiation as previously. Both corneal reflexes were present and equal. A repetition of the alcohol injection again rendered the patient free from pain. However, two months later she developed vertigo, nausea and vomiting. Examinations of the ears, nose and sinuses were negative. About a year after the second alcohol injection the facial pain recurred, although there was still anesthesia in the first and second branches of the right trigeminal nerve. Because of the history of an acute Ménière's syndrome and persistent anesthesia in the affected branches, exploration of the posterior fossa was advised with the possibility of an acoustic neuroma in mind. When the cerebellum was elevated, the fifth, seventh and eighth nerves were found stretched over a tumor mass the size of a large hazelnut. This was removed piecemeal and the nerves were left intact. The microscopic examination revealed acoustic neurofibroma. Recovery was uneventful. The patient has been free from pain and there has been a gradual return of the function of the facial nerve, so that she can close the eyelids and the nasolabial fold is present, but there is a drawing of the angle of the mouth on smiling and she is unable to whistle. Examination sixteen months after the operation revealed complete return of sensation over the right side of the face. The authors think that, when there is evidence of compression of the trigeminal sensory root and neighborhood signs such as diminished or absent corneal reflex, Ménière's syndrome and loss of hearing accompanied by ataxia and incoordination of the extremities, it is well to consider the posterior approach to the sensory root. They stress that the more simple methods of inhalation of trichlorethylene and peripheral or deep alcoholic injection are not sufficient to produce permanent relief from pain, and they warn that there have been insufficient grounds to justify the sense of security which has been felt by advocates of the transtemporal approach to the posterior sensory root of the trigeminal nerve.

Journal of Nervous and Mental Disease, New York

92:281-428 (Sept.) 1940

Localized Nonsuppurative Encephalitis Secondary to Infections of Temporal Bone and Paranasal Sinuses: Report of Four Cases. J. C. Yaskin, Philadelphia.—p. 281.
Metrazol Remission in Severe Obsession Compulsion Neurosis of Five Years' Duration. M. Zeifert, Brooklyn.—p. 290.
Tonic Neck Reflexes and Tonic Fits Following Vascular Lesions in Brain Stem: Two Cases. A. B. King, Baltimore.—p. 302.
Observations and Results Obtained in Hypoglycemic Treatment of Schizophrenia. Anna R. Coyne, Washington, D. C.—p. 309.
Threat of Mental Disease. I. S. Wile, New York.—p. 323.
Adipositas Cerebralis and Emaciatio Cerebralis. A. Gordon, Philadelphia.—p. 342.
Meningeal Permeability and Metrazol Therapy. B. Skorodin, M. L. Fisher, L. Schlan, Manteno, Ill.; S. Maurer and H. O. Wiles, Chicago.—p. 348.

Journal of Pediatrics, St. Louis

17:279-422 (Sept.) 1940

Physical Complaints Without Organic Basis. Mary I. Preston, San Francisco.—p. 279.
*Nonspecific Mesenteric Lymphadenitis: Report of Fifty-Eight Cases. I. P. Sobel and D. Stetten, New York.—p. 305.
*Treatment of Cryptorchism with Chorionic Gonadotropic Hormone and Male Sex Hormone. C. Zelson and E. Steinitz, New York.—p. 315.
Statistical Study of Otitis Media in Children. G. Heller, Englewood, N. J.—p. 322.
Build Variations in Adolescent Girls. Leona M. Bayer, San Francisco.—p. 331.
Weight and Menses in Adolescent Girls, with Special Reference to Build. Leona M. Bayer, San Francisco.—p. 345.
*Method of Diagnosing Idiopathic Epilepsy. F. C. DeLorenzo, Newark, N. J.—p. 355.
Significance of Routine Nose and Throat Cultures for Diphtheria on Pediatric Admissions. C. M. Witzberger and L. B. Slobody, New York.—p. 360.
Bacillary Dysentery Acquired at Birth. M. Greenberg, S. Frant and Rebecca Shapiro, New York.—p. 363.
Study of Incidence of Illness Among Institutional Children. H. E. Thelander, San Francisco.—p. 367.
Foreign Body in Heart: Case Report. C. Petrillo, New Haven, Conn.—p. 378.

Nonspecific Mesenteric Lymphadenitis.—Sobel and Stetten report fifty-eight proved cases of mesenteric lymphadenitis. Forty-five of the patients were less than 15 years of age. There was no apparent correlation between the occurrence of the lymphadenitis and the season of the year. Thirty patients had had one or more similar attacks of definite abdominal pain previous to the one for which they were operated on; eleven had not had a previous attack of abdominal pain, and its occurrence or absence was not mentioned in the history of seventeen. The attacks were frequently accompanied by fever and/or vomiting. Definite abdominal tenderness was elicited in fifty-four of the fifty-eight patients. Spasm or rigidity was present in the right iliac region of twenty-seven patients. The blood counts were not remarkable; usually a moderate leukocytosis and polynucleosis were present. There was one death, a boy 8½ years of age dying of bronchopneumonia three days postoperatively. Every other patient recovered after an uneventful convalescence. At operation an excess of free turbid, straw-colored or serosanguineous peritoneal fluid was found in twenty-two cases. No growth was obtained in the five cases in which this fluid was cultured. The appendix was either grossly normal, thickened or slightly or moderately injected but never acutely inflamed. Evidence of previous infection was shown by definite appendicular adhesions in twenty-three cases. The pathologic diagnoses made after microscopic examination of the appendix were chronic productive appendicitis, chronic catarrhal appendicitis, lymphoid hyperplasia of the appendix, chronic recurrent appendicitis and a few instances of acute and chronic catarrhal appendicitis. At operation the condition of the mesenteric nodes conformed in every case to the classic picture of acute nonsuppurative mesenteric lymphadenitis. Appendectomy was performed in every case.

Twenty patients were examined from one month to six years and seven months after operation. Eleven of the twenty patients have had one or more attacks of abdominal pain similar in nature but not in severity to the one for which operation was performed. The attacks did not incapacitate the patient nor did their severity cause him to seek medical aid. The authors believe that they tend to abate and disappear in the course of the years and therefore advise operation not merely for diagnosis but also for appendectomy, as they share the general opinion that in some still unexplained way the removal of the appendix favorably affects the course of the disease. None of the numerous etiologic theories advanced for mesenteric lymphadenitis have been adequately proved. The presence of adhesions of the appendix would seem to be a more definite criterion of appendical involvement than microscopic alterations alone, since adhesions of this organ certainly indicate the occurrence of previous disease. The authors believe that mesenteric lymphadenitis is even more common than heretofore supposed; it is without doubt frequently overlooked at operations performed as a result of the unavoidably incorrect diagnosis of acute appendicitis. The clinical picture, which mimics acute appendicitis so closely that laparotomy is performed in children, adolescents and young adults, is frequently caused by an overlooked mesenteric lymphadenitis. Definite though slight pathologic alterations in the appendix are generally found in this syndrome. If at appendectomy the condition of the appendix does not fully and adequately explain the clinical picture, careful exploration for mesenteric lymphadenitis should be performed. Frequently the surgeon will be rewarded by finding a mass of inflamed nodes as the true cause of the symptoms and signs.

Chorionic Gonadotropin and Testosterone Propionate for Cryptorchism.—Zelson and Steinitz gave male and female hormone preparations concurrently to seventeen boys with cryptorchism. The ages of the boys ranged between 7½ and 12½ years and all of them had been seen previously at intervals for several months. Two of the boys had bilateral cryptorchism. Treatment consisted of three weekly intramuscular injections of chorionic gonadotropin and testosterone propionate. Four boys were given 500 units of chorionic gonadotropin and 5 mg. of testosterone propionate and thirteen 500 units of chorionic gonadotropin and 10 mg. of testosterone propionate. Treatment extended over a period of from three to twelve weeks. Complete descent occurred in nine (53 per cent). Four of these had been treated previously without satisfactory results. There was no effect on the cryptorchism in the other eight children. In all but three the penis and scrotum became larger. Growth of pubic hair occurred in eight, in three an inguinal hernia became evident, the undescended testicle became smaller in three and in one bilateral case both testicles descended and grew larger. The authors believe that treatment should be instituted before 10 years of age, as too long a delay in the descent may prevent the normal development of the testicle. In consideration of the expense and in order to avoid any unnecessary premature sexual stimulation they suggest that first a course of treatment with chorionic gonadotropin be given, from 300 to 500 rat units three times a week up to a total of 10,000 units, and if no response results a combined course of treatment should then be given after a rest period of from one to three months. If still no satisfactory result is obtained the cryptorchism should be treated surgically.

Diagnosis of Idiopathic Epilepsy.—DeLorenzo employed the antidiuretic and superhydration test introduced by McQuarrie as a diagnostic test in seventeen cases of convulsions. The procedure is as follows: 1. A diet of 40 per cent cream and dextrose in antiketogenic proportions is given every six hours. 2. From 2 to 5 cc. of water per kilogram of body weight and an amount of pitressin sufficient to prevent diuresis are given at intervals of three to four hours. 3. The patient is weighed every six hours after the bladder is emptied and before food or water is taken. 4. A complete record is kept on a chart of the food, water and pitressin given, and of the weight and urine. 5. A preliminary period of dehydration facilitates the occurrence of seizures. 6. The procedure is stopped when convulsions, symptoms of water intoxication (abdominal cramps, excessive vomiting and headache) or symptoms of pitressin toxicity occur or when it is evident that convulsions will not occur. All the requirements of the test must be fulfilled and kept under strict surveillance to prevent mistakes and frequent failure. Two of the seventeen patients with convulsions had lead encephalopathy, one had convulsions following head trauma, three were less than 3 years of age, one had convulsions associated with an infection and pyrexia, one was a case of petit mal and the others had been diagnosed as idiopathic epilepsy. With the exception of two patients, all of the patients with a diagnosis of idiopathic epilepsy reacted positively to the McQuarrie test. Encephalograms done subsequent to the positive tests were negative. Of the two failures one was of a patient with hypoglycemia and in one the pitressin did not have the usual antidiuretic effect. The test was repeated a month later for the last patient and proved positive. Seventeen normal children who served as controls reacted negatively to the McQuarrie test. The child whose convulsions seemed to be precipitated by head trauma gave a negative reaction to the test; subsequently an encephalogram indicated enlargement of the left ventricle. The other patients with convulsions and the patient with petit mal reacted negatively to the test. It is difficult to determine the true significance of the test from such a small group of patients, but the authors feel that there is sufficient evidence to employ it as an aid in differentiating idiopathic epilepsy from other conditions simulating it. Negative tests will often lead to further investigation and the discovery of an entity which may be treated satisfactorily. They suggest that in idiopathic epilepsy there may be a disturbance in the normal physiologic mechanism in the cortical cell membranes and that any anatomic pathologic change found in these cases may have been the result, rather than the cause, of the disease.

Medical Annals of District of Columbia, Washington

9:291-332 (Sept.) 1940

*Treatment of Delirium Tremens and Acute Alcoholic Hallucinosis. I. J. Silverman, Washington.—p. 291.
History of Tobacco. H. G. Hadley, Washington.—p. 298.
Fads and Fallacies in Diagnosis and Treatment of Hypertension. W. M. Yater, Washington.—p. 300.
Venous Ligation in Treatment of Superficial Thrombophlebitis: Report of Case. R. K. Hollingsworth, Washington.—p. 304.
Staphylococcic Septicemia Treated with Staphylococcus Antitoxin: Report of Case. W. W. Sager, O. B. Hunter and G. Tolstoi, Washington.—p. 306.

Treatment of Delirium Tremens and Acute Alcoholic Hallucinosis.—Silverman states that after Feb. 8, 1939, the treatment for alcoholic hallucinosis at the Gallinger Municipal Hospital has been to give hypertonic sodium chloride solution intravenously. Injections of 100 cc. of a 5 per cent solution and later 150 cc. were given twice a day, with sometimes an initial dose of 200 or 250 cc. There were no deaths in 1939 among the uncomplicated cases in which the treatment was given for at least two consecutive days. As a minimal standard for the present it is suggested that when a patient does not live for forty-eight hours the treatment has not been put into proper effect. The complicated cases showed a definite diminution of mortality. There were only four deaths among uncomplicated cases in 1939 after February 8 and in none of these was the treatment given, for one reason or another. There were sixteen deaths among the complicated cases. In the first six months of 1940 one patient died in whom a complication was not definitely established. This patient was in the hospital less than forty-eight hours and had insufficient saline solution. During this six months 327 patients were given the saline treatment. The effect of the hypertonic saline solution treatment is striking in that patients soon become quiet and the period of noisy delirium is usually cut down to an hour or at the most a few hours. It appears that instances of delirium tremens have been prevented and others aborted. The patients first become quiet, then coherent and rational, often losing their hallucinations on the first or second day. If pyrexia has ensued, more rigorous treatment must be pursued. When patients are physically fit they are often sent to the hydrotherapy department for hot cabinets and the Scotch douche to complete the treatment. Vitamins are used sometimes. The specific treatment does not exclude other therapeutic measures. The technic of the intravenous medication is simple. If accidental leakage should occur at the site of injection, no harmful results accrue. Local swelling due to the attraction of fluid from surrounding parts and the initial stinging subsides as the fluid accumulates. No sloughing of

tissue has occurred and venous thrombosis rarely, causing less trouble than with 50 per cent dextrose. There were only two reactions such as occur after parenteral isotonic saline or dextrose solution among the 5,000 or 6,000 injections. The rate of flow is best controlled by the height of the cylinder rather than by the clamp. Using a 20 gage needle, it usually takes from ten or fifteen minutes for 150 cc. of the solution. The apparatus should be watched throughout the injection as the needle may shift, especially if there has been some leakage with its consequent swelling and distortion of tissues. Alcoholic psychosis is frequently complicated by other conditions and such patients should not be denied the benefits of hypertonic saline solution together with such treatment as is indicated for the complication. The sodium chloride treatment decreases the incidence of lobar pneumonia and the chances of patients with complications are improved. Spinal puncture is unnecessary, sometimes harmful and at times dangerous.

Medicine, Baltimore

19:329-440 (Sept.) 1940

Plasma Prothrombin: Vitamin K. K. M. Brinkhous, Iowa City.—p. 329.
Rheumatic Heart Disease: Pathogenesis and Etiology in Their Relation to Therapy and Prophylaxis. H. F. Swift, New York.—p. 417.

New England Journal of Medicine, Boston

223:439-480 (Sept. 19) 1940

Clinical Manifestations of Primary Syphilis. F. M. Thurmon, Boston.—p. 439.
Interpretation and Reliability of Reports of Serologic Tests for Syphilis. T. B. Mallory, Boston.—p. 441.
Detection and Treatment of Cardiovascular Syphilis. H. L. Blumgart, Boston.—p. 443.
Early Clinical and Laboratory Manifestations of Syphilis of Central Nervous System. H. H. Merritt, Boston.—p. 446.
Public Health Aspects of Syphilis as It Concerns the General Practitioner. T. Parran, Washington, D. C.—p. 450.
Nonsurgical Conditions Simulating Acute Appendicitis in Children. H. L. Heyl, Boston.—p. 454.
Cancer. G. W. Taylor, Boston.—p. 461.

New York State Journal of Medicine, New York

40:1347-1418 (Sept. 15) 1940

Observations on Prolonged Human Refrigeration. T. Fay, Philadelphia.—p. 1351.
Refrigeration in Cancer: Pathologic Observations in 100 Advanced Cases. L. W. Smith, Philadelphia.—p. 1355.
Syphilis in Industry. E. D. Osborne, H. L. Traenkle and F. A. Dolce, Buffalo.—p. 1362.
Outbreak of Typhoid Fever Associated with a Trailer Camp. P. A. Lembcke, Rochester, and P. J. Rafle, Syracuse.—p. 1371.
Incipient Psychoses and the General Practitioner. B. Liber, New York.—p. 1375.
The Premature Infant: Statistical Study. M. B. Einhorn, Albany.—p. 1380.

Texas State Journal of Medicine, Forth Worth

36:343-400 (Sept.) 1940

Infectious Mononucleosis. R. R. Kracke, Emory University, Ga.—p. 348.
Treatment of Pneumonia with Sulfanilamide, Sulfapyridine and Its Allied Compounds. W. G. Reddick, Dallas.—p. 354.
*Hemochromatosis: Report of Six Cases. J. C. Cain, Galveston.—p. 356.
Management of Premature Infants. G. T. O'Byrne, Corpus Christi.—p. 363.
Occult Syphilis. W. A. Clark and D. T. Gandy, Houston.—p. 367.
Selective Treatment of Cancer of Larynx. J. H. Foster, Houston.—p. 370.
Evaluation of Parenteral Fluid Therapy. J. W. Tottenham, Fort Worth.—p. 373.
The Climacteric. J. H. McLean, Fort Worth.—p. 377.
Endocrines in Gynecology and Obstetrics, with Special Reference to Stilbestrol in Treatment of Uterine Bleeding: Original Research on Menstruation. K. J. Karnaky, Houston.—p. 379.
Evaluation of Iodized Oil as Therapeutic Agent in Treatment of Bronchial Asthma. G. J. Seibold, Wichita Falls.—p. 386.

Hemochromatosis.—Cain reports six cases of hemochromatosis encountered at postmortem examination in the pathology department of the University of Texas. None were diagnosed before death. The oldest case dates back to August 1917. Hemochromatosis was a contributing factor and not the cause of death. The onset of the symptoms was slow in five of the six cases. Diabetes was present in only two. Polyuria was present in these and also in one other case, suggesting an early subclinical diabetes. Abdominal symptoms were present in all except one. They probably resulted from the terminal conditions rather than from the hemochromatosis and therefore were of little value. The histories of two patients showed an excessive use of alcohol and a moderate, and possibly an excessive, use by two others. Five of the six patients definitely mentioned weakness as a predominant symptom. The weakness was progressive and could not be attributed entirely to the terminal disease. Splenic enlargement was present in three and definite cirrhosis of the liver in five. Only one patient showed gonadal hypoplasia and four made no mention of the genitalia or of impotence. The testicles of one patient were grossly normal. The heart was enlarged in four, and a generalized lymphadenopathy was present in three and marked jaundice in three. Pigmentation of the skin is one of the characteristic signs of hemochromatosis and it usually attracts attention first. Unfortunately, pigmentation may be completely absent. Two of the author's patients showed pigmentation, it was absent in two, both of whom were Negroes, and no mention of the skin of two was made. No microscopic sections were made of the skin in any of the cases. Although one of the Negroes showed no abnormal pigmentation of the skin, there were pigmented areas in the mucosa of the gums and palate. The ages of the patients at death were respectively 75, 52, 48, 50, 73 and 39 years. The death of the 39 year old patient was untimely. It was due to traumatic rupture of the urinary bladder. All the patients were men. Five of them lived for less than eleven months after the onset of symptoms. The other patient lived for nine years. One patient died of pernicious anemia, two of carcinoma of the prostate, one of arteriosclerotic heart disease and one of a terminal Staphylococcus albus septicemia complicating a severe diabetes mellitus. Grossly the skin may vary from the slate gray color of argyria to that seen in Addison's disease. It usually has a definite bronzed appearence. Microscopically there are deposits of hemosiderin in the corium and melanin deposits in the stratum malpighii. Microscopically the changes in the liver are those of cirrhosis and pigmentation. Iron stains show enormous amounts of hemosiderin in the hepatic cells. The pigment usually destroys the cell, and fibrous tissue replaces it. Hemofuscin is also present, but it is deposited chiefly about the adventitial coat of the blood vessels. The microscopic appearance of the pancreas, which was involved in five cases, is much like that of the liver. The appearance of the diabetes mellitus depends on destruction of the islet cells, which may occur early or late. Deposits of hemosiderin and hemofuscin were present in all six spleens. Iron stains showed most of the hemosiderin to be deposited about the trabeculae and the blood vessels. There was some increase in the fibrous tissue. Of the lymph nodes the portal and pancreatic are most commonly involved but none are immune. Iron stains show hemosiderin in large amounts, which crowds out the normal structures and causes necrosis and atrophy with replacement fibrosis. The adrenals were involved in four cases, showing deposits of hemosiderin in the zona glomerulosa with little excess of fibrous tissue. Any organ in the body may be involved.

Virginia Medical Monthly, Richmond

67:593-654 (Oct.) 1940

Psychiatry and the Courts: Some Attitudes and Their Reasons. W. Overholser, Washington, D. C.—p. 593.
Lobar Pneumonia Among the Mentally Ill. L. Kolipinski, Petersburg.—p. 599.
Mental Hygiene. H. C. Henry, Richmond.—p. 600.
Experiences with Insulin and Metrazol in Treatment of Eighty Psychotic Patients. J. C. Palmer and J. P. King, Radford.—p. 603.
Some Comments on Intramural and Extramural Activities of a Modern State Hospital. J. E. Barrett, Marion.—p. 605.
Mental Hygiene and Psychiatry. P. H. Drewry Jr., Richmond.—p. 609.
The First 100 New Admissions to the Petersburg State Colony. D. L. Harrell Jr., Petersburg.—p. 613.
Acute Surgical Lesions of Abdomen. I. Abell, Louisville, Ky.—p. 615.
Management of Urinary Infections in Children. R. M. Tyson, Philadelphia.—p. 623.
Endocrinology Briefs: Conclusion: Miscellaneous Glands. J. P. Lynch, Richmond.—p. 629.
Acute Traumatic Arterial Thrombosis of Extremities. E. L. Lowenberg, Norfolk.—p. 630.
Vinethene. W. M. Whitehead and C. C. Carter, Juneau, Alaska.—p. 635.
Low Back Pain. H. W. Kinderman, Palmer.—p. 637.
Anesthesia as Used in the Small Hospital. J. T. Rountree and Nora Whitever, Woodstock.—p. 638.
Bilateral Dermoid Cysts Complicating Normal Pregnancy: Case Report. W. R. Southward Jr., Richmond.—p. 641.

FOREIGN

An asterisk (*) before a title indicates that the article is abstracted below. Single case reports and trials of new drugs are usually omitted.

Archives of Disease in Childhood, London

15:129-200 (Sept.) 1940

Generalized Angiomatosis Presenting Clinical Characteristics of Storage Reticulosis, with Some Observations on Reticulo-Endothelioses. L. G. Parsons and J. H. Ebbs.—p. 129.
Sternal Marrow Puncture in Children. M. Diwany.—p. 159.
Simple Specific Precipitation Test for Cow's Milk. A. Gnosspelius.—p. 171.
Rheumatic Heart Disease in Identical Twins. C. B. Perry.—p. 177.
Incidence of Four Main Blood Groups in Rheumatic Heart Disease. G. R. Maxted.—p. 181.
*Carbohydrate Tolerance in Hypothyroidism and Hyperthyroidism. T. Crawford.—p. 184.

Carbohydrate Tolerance in Hypothyroidism and Hyperthyroidism.—Crawford tried to determine the changes of carbohydrate tolerance of six hypothyroid and two hyperthyroid children and to explain the manner in which these changes are produced. Children are suitable subjects for such a study, as their lesions usually occur in a pure form so that the metabolic disturbances can be assumed to depend on the altered state of the thyroid secretion. In adult myxedematous subjects conflicting factors (arteriosclerosis and postmenopausal polyglandular disturbances) are frequently present and tend to distort the results of metabolic tests and render their interpretation more difficult. Two tests have been employed to gage carbohydrate tolerance: the oral dextrose tolerance test and the intravenous dextrose tolerance test. In cretinism the fasting blood sugar level was frequently subnormal and tended to rise under treatment with thyroid preparations. Results of the oral dextrose tests were irregular. Low curves were frequent, but normal and high curves also occurred. Intravenous dextrose tolerance tests revealed impairment of dextrose tolerance. This is in contrast to the usual assertion that tolerance to carbohydrate is increased in hypothyroidism. Under treatment all the curves tended to approach normal; but if excessive dosage of thyroid was given impairment of tolerance reappeared in the form of high oral and delayed intravenous curves. Of the two cases of hyperthyroidism studied one showed a high oral curve with glycosuria and the other had an oral curve within normal limits. Both exhibited impaired tolerance to intravenous dextrose. The author concludes that the impaired carbohydrate tolerance in hypothyroidism is due to a general slowing of the processes of catabolism and possibly to a diminution in the storage rate of carbohydrate. The low oral blood sugar curve is due to slow absorption of the carbohydrate from the intestine. The impaired carbohydrate tolerance of hyperthyroidism is traceable to defective glycogen formation. This probably results from the excessive secretion of thyroxine causing an increased liberation of epinephrine, which in its turn mobilizes hepatic glycogen and inhibits its reformation.

British Journal of Radiology, London

13:293-324 (Sept.) 1940

Supplementary X-Ray Treatment for Carcinoma of Cervix Uteri in Relation to Direction of Spread of Disease. A. G. C. Taylor.—p. 293.
Dose Measurement in Interstitial Radium Therapy. A. A. D. La Touche and F. W. Spiers.—p. 314.
Percentage Depth Doses in Low Voltage X-Ray Therapy. W. J. Meredith.—p. 320.
Nomogram for Determination of Lead Protection Against High Voltage X-Rays. W. Binks.—p. 322.

Indian Medical Gazette, Calcutta

75:385-448 (July) 1940

Observations on Some Unusual Toxic Effects of Sulfanilamide Therapy. M. N. De and N. R. Konar.—p. 385.
Treatment of Opium Addiction with Lecithin and Glucose and Its Effects on Abstinence Symptoms. R. N. Chopra and G. S. Chopra.—p. 388.
Radiologic Appearances of Heart and Lungs in Cases of Hypertensive Heart Failure. R. J. Vakil.—p. 392.
Nonepidemic Typhus Fever in the Civil Population of Bangalore (Civil and Military Station). L. R. Sharma.—p. 398.
Common Cold Responsible for Grave Suspicion of Rabies in Dog and Discussion on Associated Considerations Including Rabies in Mongooses and Bats. S. D. S. Greval and M. J. Nicholas.—p. 401.
Hormones in Menstruation and Pregnancy: Physiology, Pathology and Treatment: Résumé. A. P. Pillay.—p. 404.

Journal of Physiology, Cambridge

98:389-502 (Sept.) 1940. **Partial Index**

Weight of Chromophore Carrier in Visual Purple Molecule. E. E. Broda, C. F. Goodeve and R. J. Lythgoe.—p. 397.
Action of Antidiuretic Principle of Posterior Pituitary Extracts on Urine Excretion of Anesthetized Animals. H. Heller.—p. 405.
Pituitary and Insulin Content of Pancreas. R. E. Haist.—p. 419.
Action of Some Amines Related to Adrenalin: Phenylallylamine, Phenylbutenylamine, Diphenylethylamine. J. A. Gunn and M. R. Gurd.—p. 424.
Relation of Heart Rate to Intracranial Pressure. O. G. Edholm.—p. 442.
Observations on Osmotic Pressure of Endolymph. P. Aldred, C. S. Hallpike and A. Ledoux.—p. 446.
Slow Vaginal Potentials. R. B. Bourdillon and O. M. Lidwell.—p. 480.
Action of Adrenalin on Uterus of Guinea Pig and Its Modification by Eserine. W. T. Agar.—p. 492.

Lancet, London

2:317-350 (Sept. 14) 1940

*Glossitis and Vitamin B_2 Complex in Pellagra, Sprue and Allied States. P. Manson-Bahr.—p. 317.
Lung Abscess Treated with Sulfanilamide. G. F. Taylor, M. Yusuf and N. L. Chitkara.—p. 320.
Postpartum Necrosis of Anterior Lobe of Pituitary. H. L. Sheehan.—p. 321.
Vertebral Injuries in Convulsion Therapy and Epilepsy. D. Blair.—p. 325.
Idiopathic Myalgia Simulating Visceral and Other Diseases. M. Gutstein-Good.—p. 326.
*Psychiatric Complications of Hypoglycemia in Children. E. W. Anderson.—p. 329.

2:351-380 (Sept. 21) 1940

*Treatment of War Wounds of Limbs: Experience in 266 Cases. S. M. Cohen and C. A. R. Schulenburg.—p. 351.
*Glossitis and Vitamin B_2 Complex in Pellagra, Sprue and Allied States. P. Manson-Bahr.—p. 356.
Mechanism of Cerebral Concussion. K. C. Dixon.—p. 360.
Intravenous Insulin in Treatment of Schizophrenia. M. Jones.—p. 361.

Glossitis and Vitamin B_2 Complex in Pellagra, Sprue and Allied States.—Manson-Bahr thinks that the aphorism "raw red tongue, raw red gut" may contain more than a grain of truth; the tongue may be regarded as a mirror of the stomach and intestine. Sprue tongue is well known, and a similar condition is seen in pellagra, pernicious anemia and idiopathic steatorrhea, which are characterized by anemia and intestinal upset. Known etiologic factors in this group of diseases include nicotinic acid and riboflavin deficiency. This paper is based on clinical observations made with the object of disentangling the etiology of tropical sprue and allied diseases. In the present state of knowledge it is impossible to envisage the exact manner in which these various syndromes are related, but it is clear that there exists some common link or chain connecting them. It has been shown that glossitis, probably of a similar nature and nonspecific to the disease, is found in pellagra, sprue, pernicious anemia, the nutritional anemias and idiopathic steatorrhea and that through this and other features, such as changes in the spinal cord, these diseases are closely related to one another. It is therefore suggested, on analogic as well as therapeutic grounds, that a deficiency of the vitamin B_2 complex, which appears to be common to all, may partially explain these phenomena. It is further suggested that nicotinic acid and riboflavin play an important part in the treatment of sprue as well as of pellagra, pernicious anemia, subacute combined degeneration of the cord and, to some extent, of idiopathic steatorrhea. It is further shown that these diseases are so closely related to one another that borderline cases are often encountered embodying the salient features of two or even more of the group. The opinion is therefore expressed that this group of diseases has as many affinities in common as there are factors involved (some of which may still be obscure) and that the clinical picture of any one of them varies with the presence or absence of any one or more of these factors.

Psychiatric Complications of Hypoglycemia in Children.—Anderson points out that hypoglycemia, whether from overdosage of insulin or from hyperplasia or neoplasm of the islets of Langerhans, or from any other cause, shows itself most in symptoms referable to the nervous system. Baker in 1939 described a prodromal stage of nervousness, sweating, pallor, tremor, sleepiness and salivation, which may pass on to diplopia, muscular twitchings of the face and limbs, hemiplegia and jacksonian or epileptiform attacks. Other disturbances

include aphasia, disorders of sensation, hemianopia or complete blindness, auditory changes and incoordination. Finally the patient may become stuporous, with various reflex changes. Psychic disturbances sometimes accompanying the neurologic signs may begin as fears, anxieties, mild depression and faulty concentration and pass on to a stage in which the speech is thick, the patient grimaces and laughs or cries without reason and is dull or garrulous, and fugues and hallucinations may result. He may finally lose consciousness and, when he recovers, have no memory of the attack. Other authors describe a wide range of disturbances, including a fall of tension in the eyeballs, akinesia and masklike stiffness of the face, hypothermia, migraine, irritability, megaphony, behavior disorders, such as stealing, personality changes, with euphoric indifference and docility, mild depression, severe agitation and catatonia with grandiose delusions, besides psychoneurotic pictures. The association of epileptiform convulsions with hypoglycemia, especially in children, has often been noted, but the available evidence shows that the relationship between them is not constant. The author reports the histories of two children with hypoglycemia who developed neuropsychiatric symptoms. In the first one the hypoglycemic coma, which developed after an excessive dose of insulin, lasted four days and the patient then showed gross generalized dementia with symptoms suggesting an extensive lesion of the left side of the brain. After fifteen months there was only partial recovery of function. The second patient was a diabetic boy who had always been sensitive to insulin. He showed a behavior disorder, which was most manifest when the blood sugar was low. While in a hypoglycemic phase he stabbed another child. After treatment he improved considerably in both intelligence and behavior. The author discusses the difficulties encountered in the treatment of diabetic children and calls attention to the possible dangers from the newer forms of insulin.

Treatment of War Wounds of Limbs.—Cohen and Schulenburg report observations on 266 cases of gunshot wounds of the extremities. Since even small bomb splinters were often found to have produced severe internal destruction of the tissues, the authors always employed general anesthesia. The routine use of evipal and pentothal sodium followed by gas and oxygen was most satisfactory. The skin surrounding the wound was cleansed with ether soap, shaved and washed with saline solution, the wound in the meantime being covered with a saline swab. Except for excision of the skin edge no extensive débridement was carried out, but the wound was laid widely open by means of several radiating incisions. No attempt was made to excise or trim muscles. In packing, care was taken that there should be uninterrupted drainage from the cellular spaces between the muscle bellies. Tendons were not sutured, but their frayed ends were removed. Bone fragments were removed only when completely detached. No suturing or trimming of divided nerves was done. The operation was always preceded by x-ray examination. Foreign bodies were removed only when readily accessible. Large splinters were always removed but not the small ones lying some distance from the entry wound. In a few cases, localized abscess formation around a foreign body subsequently necessitated incision. Petrolatum gauze was used for packing. A thin layer of sterilized plaster wool was placed over the wound; the rest of the plaster was skin tight. Altogether eighty-four cases due to gunshot wounds were treated in plaster. Of these, fifty-four cases were compound fractures, thirty-six being of the upper extremity and eighteen of the lower. All compound fractures were treated in closed plasters. Reduction was obtained only by manual traction and maintained while the plaster was being applied. There were 207 wounds involving only the soft tissues, of which thirty were treated in plaster. These thirty were extensive wounds of the limbs, and the same principles were applied as if an associated fracture had been present. All patients operated on received a course of chemotherapy. An initial dose of three tablets of sulfanilamide was followed by two tablets every four hours for forty-eight hours, and then one tablet every four hours for forty-eight hours, making a total dosage of 19.5 Gm. in four days. The nursing staff was told that no dose was to be omitted, even at night. No toxic effects were observed from this dosage. There were no deaths, only one limb was amputated, healing has been rapid and uneventful, and after ten weeks the general condition of all patients is excellent. The value of the closed plaster method both for fractures and for injuries of the soft tissues is thus confirmed.

Medical Journal of Australia, Sydney

2:209-228 (Sept. 7) 1940

The Citadel. C. H. Fitts.—p. 211.
Value of Preoperative Estimations of Serum Protein Concentration in Gastric Surgery. J. Devine.—p. 214.

Helvetica Medica Acta, Basel

7:115-224 (Aug.) 1940

*Acute Necrosis of Pancreas and Its Treatment. R. Weyeneth and F. Wassmer.—p. 115.
*Aspects of Bronchial Carcinoma. A. Weigl.—p. 142.
Influence of Vitamin C on Labor Pains. W. Käppeli.—p. 169.
Electrocoagulation of Chronic Metritis of Neck of Uterus. T. Marti.—p. 197.

Acute Necrosis of Pancreas.—Weyeneth and Wassmer suggest that pancreatitis should be considered in the presence of an acute abdominal syndrome if the history reveals an old or recent disorder of the hepatobiliary or gastroduodenal tracts, and particularly if the patient is obese or addicted to alcohol. The face of the patient with pancreatitis is generally pale or somewhat cyanotic. If the biliary passages are involved, the appearance is subicteric. The patient sweats profusely and complains of vertigo and of trembling of the fingers. Vomiting is frequent, at first of food and later of bile. The abdomen often shows supra-umbilical distention. The pain is localized in the left half of the epigastrium and is extremely violent. Although of considerable diagnostic significance, the symptom pain is not sufficient to establish the diagnosis. Modern laboratory methods are absolutely essential. Examination of the feces permits evaluation of the external secretory function of the pancreas, steatorrhea indicating insufficiency. The detection of deviation of the pancreatic ferments is of primary importance. Wohlgemuth's method is used for the determination of the diastase content of the urine. The authors prefer Baltzer's microdetermination for the examination of the blood serum, the modification of Wohlgemuth's method (for blood instead of urine) not being sufficiently precise. The authors regard acute pancreatic necrosis as a manifestation of autodigestion initiated by the activation of trypsin and manifested by necrosis and hemorrhage, while the lipase subsequently produces fat necrosis. The frequency of this pancreatic disorder in the presence of lithiasis indicates the important role of bile in the development of pancreatitis. The histories of twenty-six cases observed at the surgical clinic of Geneva are presented. Five of the patients were treated conservatively and have improved. Of the twenty subjected to surgical treatment, seventeen died. They were operated on during the acute phase. The three patients who survived the operation had been operated on about two weeks after the onset. The authors are convinced that the operation, instead of exerting a favorable influence on the condition, leads to toxic shock. The treatment employed with success in five of the cases was as follows: 1. Strict abstinence from food and liquids. 2. Restriction of chlorides for forty-eight hours; this is done despite the hypochloremia in order to diminish the pancreatic edema. 3. Daily administration of from 1 to 2 liters of a 10 per cent solution of dextrose by intravenous infusion and from 15 to 30 units of insulin. 4. Large doses of atropine to diminish the pancreatic secretion. 5. Relief of the intestine by repeated enemas, eventually with a 1 per cent solution of quinine. 6. Ice bag to the epigastric region and morphine. After the acute phase has passed (in four or six days), fruit juices and carbohydrates can be given. Later the biliary passages can be examined and if necessary, an operation on the biliary tract may be performed in order to prevent recurrences.

Bronchial Carcinoma.—Weigl discusses the present status of bronchial carcinoma and presents observations on thirty-three cases observed in his clinic. The fact that thirty of these were in men proves again the high incidence in men. The author cites evidence indicating that, besides hereditary factors, certain industrial hazards (work in dye factories or exposure to silica dust) and smoking play a part in the etiology. In his own

cases no connection could be established between the bronchial carcinoma and occupation. The majority were in the fifth or sixth decade of life. Thirty-three per cent of the patients had a family history of carcinoma and 50 per cent had had pulmonary or pleural disorders such as pneumonia, exudative pleurisy, influenza, chronic smokers' bronchitis and apical tuberculosis. A considerable number presented thoracic or spinal anomalies. The onset was insidious in 70 per cent and acute in 30. In cases in which the onset was insidious the complaints were nearly always the same and quite characteristic. There was an increasing, irritative cough; the sputum was at first mucopurulent and later tinged with blood. Raspberry jelly sputum was rarely encountered. In approximately 75 per cent increased temperatures caused by stagnation bronchitis and bronchopneumonic attacks were observed for long periods. Many did not ask medical aid until severe symptoms such as hemoptysis, dyspnea and increasing hoarseness appeared and the carcinoma was already in an advanced stage. In these cases cachexia was generally present. The muscular tonus and cutaneous turgor were greatly reduced. Some patients had a grayish pallor, others presented subicterus or cyanosis. A pulmonary process was indicated by severe dyspnea, retraction of the thorax and lag of the involved side in respiration. Cardiac involvement was often observed, usually in the form of hypertrophy and dilatation of the right chambers. Metastases were demonstrable in about 50 per cent. The localization of the tumor was rarely possible by physical methods. X-ray examination assumes a decisive role in the diagnosis. The following roentgenologic appearances were observed: partial atelectasis, hilus tumor, greatly increased density of the hilus, solitary tumor shadows usually with mantle pneumonia, effusion and seropneumothorax. Secondary roentgenologic signs were elevation of the diaphragm on the side of the tumor, dislocation of the heart, trachea and mediastinum, sinus effusion, induration, hilar lymphoma and pulmonary shrinkage. Bronchography was performed in four cases, tomography proved valuable in three, and tracheobronchoscopy was done in seven. Exploratory excision made possible a correct histologic diagnosis in only one case. The sedimentation speed was nearly always increased. About 50 per cent of the patients had a mild leukocytosis. In seven of the patients the treatment consisted of Coutard's protracted fractional roentgen irradiation. The others were subjected to symptomatic treatment. The roentgen irradiation produced temporary improvement, reduction of pain and perhaps a slight prolongation of life. The average survival was less than a year. Treatment is generally unsuccessful. Some success is being reported with lobectomy or pneumectomy.

Acta Pædiatrica, Stockholm

27:403-516 (June 30) 1940

*Studies on Prognosis of Children Delivered by Cesarean Section. O. Brandberg.—p. 403.
Investigation of Ascorbic Acid Excretion in Urine of Healthy and Febrile Children. G. Görtz.—p. 429.
State of Nutrition of So-Called Underweight School Children. A. Ruotsalainen.—p. 437.
Follow-Up Examination of Children with Positive Tuberculin Reaction: Three Cases of Biologically Cured Tuberculosis Infection. P. Rosenberg.—p. 452.
*Albers-Schönberg's Disease (Marble Bones). S. van Creveld and N. I. Heybroek.—p. 462.
Postvaccinal Encephalitis in Child Formerly Suffering from Myasthenia Gravis Pseudoparalytica. L. Grönlund.—p. 495.
Hypophysial Nanism Resulting from Craniopharyngioma. E. Gjörup.—p. 508.

Prognosis for Children Delivered by Cesarean Section.—The material studied by Brandberg includes children delivered by classic or by vaginal cesarean section during the years from 1918 to 1939. It includes 367 cases, 292 of classic cesarean section and seventy-five of vaginal cesarean section. The immediate mortality (born dead or dying within two hours after birth) amounted to 7.5 per cent in the case of classic cesarean section and 54.7 per cent in the case of vaginal section. The mortality between two hours and one month of life was 4.82 per cent for those delivered by the classic cesarean section and 23.5 per cent for those delivered by the vaginal cesarean section. The figure 4.82 per cent does not exceed the figure for the newborn in general. Among the prematurely delivered the mortality was 66⅔ per cent; among the full-term infants it was 9.31 per cent. As was to be expected, the mortality of the infants was low or practically zero in those cases in which the intervention was made to deliver a living child, because dystocia was foreseen (contracted pelvis, tumors or advanced age of a primipara); it was higher in the cases in which it was performed on account of danger to the life of the mother (hemorrhages of placenta praevia, premature detachment of the placenta, eclampsia and so on). Apnea and asphyxia are not unusually frequent in children delivered by cesarean section. Mental and neurologic defects were observed in 5 per cent of the reexamined children. However, not all of those defects could be traced to an obstetric trauma; in about half of these cases other causes must be assumed. The frequency of cerebral injuries traceable to a trauma intra partum is 1.76 per cent in the classic cesarean section and 12.5 per cent in vaginal cesarean section; however, the median error is so high that these figures are not reliable. The author is of the opinion that more cerebral injuries would be found if all infants who die shortly after delivery should be subjected to postmortem examination. This was not done in the material analyzed here.

Marble Bone Disease of Albers-Schönberg.—Van Creveld and Heybroek describe two cases of marble bone disease or of osteosclerosis fragilis generalisata. The first patient was a newborn baby who died on the ninth day of life; the second was a girl who had been blind from birth and in whom the disease was discovered at the age of 4½ years. Although the literature contains reports indicating that familial occurrence may be expected in about 40 per cent of the cases, it was not hereditary in either of the two children whose histories are reported here. The older child, however, had consanguineous parents and the newborn child had two siblings in whom Alban Köhler's disease was diagnosed. The older child showed a retardation in physical development, but the mental development was normal. Her head was enlarged, which is probably due to the thickening of the cranial bones as well as to a slight hydrocephalus. The bilateral atrophy of the optic nerve, which caused blindness, was the first manifestation of the disease. The child had been subject to repeated bone fractures even after slight trauma. The decrease in elasticity is regarded as the chief cause of this tendency to fractures. The thickening of the cortical and spongy tissue was not general in this patient, though in some typical places (skull, ribs, vertebrae and femurs) the osteosclerosis was so pronounced that macroscopic inspection disclosed no difference between the cortex and the spongiosa. The characteristic clubbing of the long bones was readily demonstrable. The presence of bands running parallel to the line of the epiphysis was noticeable especially in the bones of the hands and feet and in the metaphyses. In the scapulas, in both ossa ilii and in the tarsal and carpal bones these bands were ring shaped. The factors responsible for the formation of these bands are unknown, but it is suggested that their existence points to periodic fluctuations in the process of sclerosis. Symptoms indicating endocrine disturbances were absent in both children. Biochemical tests revealed no signs of hyperparathyroidism. The blood of both children had normal values for calcium, inorganic phosphorus and magnesium and in the older child the ultrafiltrable calcium and inorganic phosphorus were likewise normal. Both children had the normal number of units for (alkaline) phosphatase. The newborn child had an abnormal blood picture characterized by an extraordinary erythroblastemia, thrombopenia, increase in the staff cells, juvenile forms and myelocytes, also increase of the mean diameter of the erythrocytes. In the older child the blood picture has been normal up to now. This absence of anemia the authors regard as an argument in favor of the conception that the bone anomalies of marble bone disease are primary and not dependent on the damage of the bone marrow. The authors review various theories suggested to explain the osteosclerosis of marble bone disease. With regard to the diagnosis they stress the atrophy of the optic nerve, the tendency to bone fractures, the malformation of the thorax, the thickening of the lower end of the femurs, the peculiar shape of the head and more or less incomplete teeth. No effective therapy is known for this disease.